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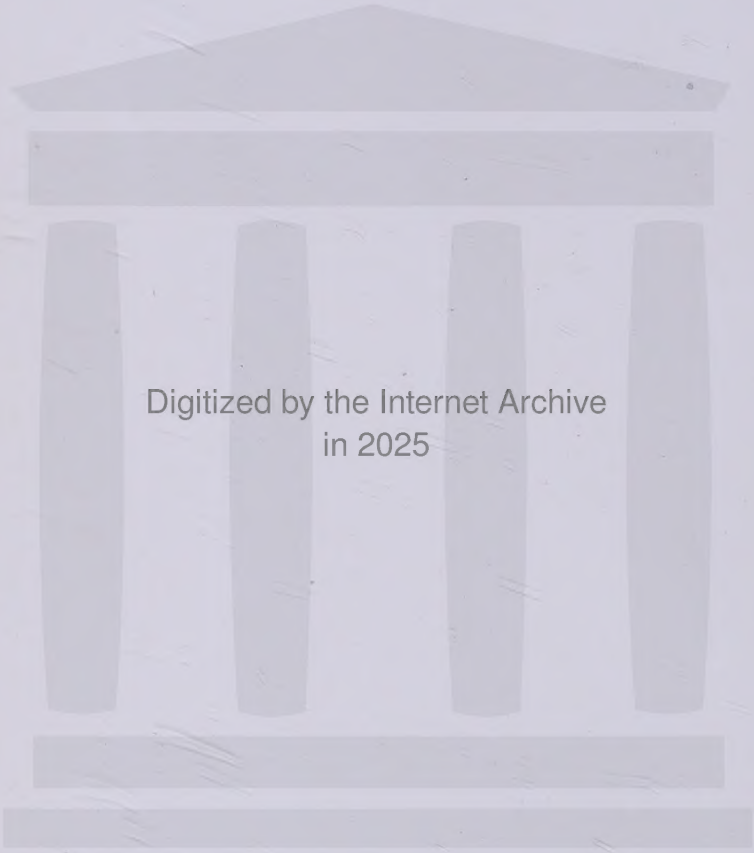
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THE FIRST FORMAL UNION OF NEURO-ANATOMY AND ART. (*Stephanus*, 1545.)

"The avulsed calvarium hangs from a tree, showing the dura mater."

TEXT-BOOK
OF
NERVOUS DISEASES

FOR THE USE OF STUDENTS AND PRACTITIONERS OF MEDICINE

BY

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EIGHTH EDITION

WITH TWO HUNDRED AND SIXTY-TWO ILLUSTRATIONS, INCLUDING FOUR PLATES IN BLACK AND COLOR

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PREFACE TO THE EIGHTH EDITION

When the first edition of my book was published twenty-three years ago there was no adequate description of the anatomy of the nervous system in the English language. Hence, feeling the fundamental importance of this subject to an understanding of neurology, I gave much space to it. This need of anatomical knowledge of the nervous system is now abundantly supplied by general and special text-books. I have, therefore, left out a good deal of this part of my text. I have retained and revised what was especially useful in diagnosis and for reference, and have also changed, added to and improved the illustrations. In this I have received help and skilled supervision from Dr. J. F. Gudernatsch, of the Anatomical Department of Cornell University.

A large part of the rest of the book has been completely remodeled and rewritten. This applies to the chapters on syphilis of the nervous system, including paresis, tabes and the serology of nervous diseases. Dr. David Kaplan, of the New York Neurological Institute, has furnished me with data on this last subject which represent large experience and bring the matter to date.

The chapter on acute anterior poliomyelitis has been rewritten by Dr. Foster Kennedy in the light of recent discoveries, and the same has been done for the chapter on epidemic cerebrospinal meningitis by Dr. H. W. Frink. I am under obligations to both these gentlemen for their excellent work.

Practically new chapters have been written on tumors of the spinal cord and tumors of the brain. Dr. Charles A. Elsberg has allowed me to use the statistics of his cases, so that I am able to present these topics from the standpoint of modern neurological surgery as well as of medicine.

The chapters on methods of examination and diagnosis and on general symptoms have been thoroughly revised and an endeavor made to include the latest data. The number of epinomic signs and syndromes of neurology has grown to a burdensome length. A descriptive list of them has been prepared by Dr. Frink.

The chapters on the psycho-neuroses have been rearranged and in parts rewritten and condensed. Neurasthenia has been pushed, as modern views demand, much more into the background. Hysteria and psychasthenia have been treated from the standpoint of descriptive rather than of "dynamic" or analytic neurology. This it seemed to me was the only way to make the subject intelligible in a work meant largely for

students and practitioners. I trust that I have, however, given a measure of justice to psycho-analysis, as it certainly has a place in the interpretation and occasionally in the therapeutics of the minor psychoses.

The subject of the disturbances of growth and metabolism and of nervous function due to glandular disorders has brought out a large literature in recent years, and this has been considered in the revision of this edition.

The subject of therapeutics is difficult to discuss effectively in a treatise that aims to be of moderate size. Methods and drugs and points of view change so rapidly that I have decided to leave out the special chapter on this subject. Treatises on electricity, massage, exercise, diet, mechanical therapy, hydrotherapy, etc., are now many and accessible.

Many new clinical and anatomical illustrations have been added. I am greatly indebted to Dr. J. B. Gere, of the Pathological Department of Cornell University Medical College, for beautiful photographs of spinal-cord and of nerve disease; to Dr. S. Wachsmann and the Montefiore Home for photographs of clinical cases; and to Dr. R. S. MacRobert, of New York Neurological Institute, for photographs illustrating methods of examination. A modest tribute has been paid to the fathers of neuro-anatomy in the reproduction and use of illustrations of the brain by Stephanus, Willis, and Ruysch. They represent anatomical illustration and art in the 16th, 17th and 18th centuries respectively.

The part of my work devoted to psychiatry will appear in a separate volume. However, I have included in the present treatise articles on the minor psychoses, as well as a chapter on paresis.

A treatise on neurology must usually be read, and studied in parts, hence that portion of the original preface has been retained which suggested to the reader a certain eclecticism of method in neurological study. The principle then set forth holds good now though the lists then given need revision.

My thanks are due to my publishers for their helpfulness and cheerful co-operation.

New York City,
Sept., 15, 1915.

FROM PREFACE TO THE FIRST EDITION

As a special text-book the present work will be used by two classes of readers, one consisting of those who simply consult it for reference in connection with their cases, the other composed of students who desire to ground themselves systematically in a knowledge of neurology. To this latter class I venture some advice as to the method they should pursue. Neurology is a difficult branch of medicine to master, nor is there any royal road to it. Still, it can be made comparatively easy if its study is undertaken in a proper and systematic way.

In using the present work, the student should first refresh his general knowledge of nervous anatomy as furnished in ordinary text-books. He should then go carefully over the anatomical descriptions here given of the general structure of the nervous system and of that of the nerves, spinal cord, and brain. A thorough knowledge of anatomy and physiology makes clinical neurology comparatively easy, and in fact reduces much of it simply to a matter of logical deduction.

The student should next master the general facts of nervous pathology, symptomatology, and etiology, for he will find common laws underlying apparently the most varying phenomena. Finally, he must begin to study the special diseases. The number of these is very great; in the present work I have described 176. Many of these are rare, and it would be wrong for the student to burden his memory with the details about them. He need know only of their existence and general physiognomy. There are, however, according to my enumeration, about 65 nervous diseases which are either very common or extremely important, and it is these that the student should master and make part of his working knowledge. Since the distribution and names of the common and rare diseases may be a useful guide, I append here a table and a list:

	Peripheral	Spinal Cord	Brain	Functional	Totals
Common and important diseases	31	13	12	10	66
Rare	56	27	16	11	110
	87	40	28	21	176

The common or important nervous diseases are:

General.—Neuritis, multiple neuritis, degeneration, neuralgia, paræsthesia (5).

Cranial Nerves.—Anosmia, optic neuritis, optic atrophy, ptosis, ophthalmoplegia, abducens palsy, headache, migraine, trigeminal neuralgia, facial spasm, facial palsy, tinnitus, vertigo, ageusia, wryneck (16).

Spinal Nerves.—Cervical neuralgia, hiccough, brachial palsies, single and combined, brachial neuralgia, intercostal neuralgia, herpes zoster, lumbar neuralgia, sciatica, leg palsies (10).

Spinal Cord.—Spina bifida, hemorrhage, pachymeningitis, leptomeningitis, poliomyelitis, transverse myelitis, acute and chronic, secondary degenerations, locomotor ataxia, the progressive muscular atrophies, bulbar palsy, muscular dystrophies, spinal irritation (13).

Brain.—Malformations, hyperæmia, pachymeningitis, leptomeningitis, simple, tuberculous, and epidemic, abscess, hemorrhage, embolism, thrombosis, children's palsies, syphilis (12).

Functional.—Epilepsy, hysteria, the tics, chorea, tetanus, neurasthenia, spermatorrhœa, exophthalmic goitre, occupation neuroses, paralysis agitans (10).

New York City, 1892.

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PART I

DISEASES OF THE NERVOUS SYSTEM

CHAPTER I

GENERAL ANATOMY, PHYSIOLOGY, AND CHEMISTRY

In studying the phenomena of life in the human body, we as physicians first learn about its normal structure and functions. We then note the new phenomena which develop when disease comes on, the causes which produce them, and the anatomical changes lying back of them; we group our facts and give the disease a name. Lastly we apply the methods by which the disorder can be expelled and future attacks prevented. In fine, we investigate our subject just as we do that of any branch of natural history. Our study divides itself, therefore, into

Normal anatomy and physiology.

Etiology, a study of the causes.

Symptomatology, a study of the morbid phenomena.

Pathology, under which we include a study of the morbid anatomy and physiology.

Diagnosis, or the method of recognizing and separating out the different groups of diseases.

Prognosis, a forecast of the future course of the malady.

Treatment and prophylaxis.

GENERAL ANATOMY

The nervous system is derived from the ectodermal germ layer of the developing ovum, and its constituents are modifications of epithelial cells. These cells in the embryo are of two kinds: neuroblasts, which develop into nerve-cells and fibres; and spongioblasts, which develop into a supporting structure called neuroglia (His).

The nervous system is composed of:

(*a*) Neurons, which form the nervous tissue proper, and are made up of nerve-cells, with their processes, one of which becomes an axis cylinder; and neuroglia.

(*b*) Accessory tissue, consisting of connective tissue, blood-vessels, lymphatics, and epithelium.

These tissues are united together to form the central nervous system, consisting of the brain and spinal cord, and the peripheral nervous system. This latter is composed of nerve-fibres, and structures attached to the terminations of the nerves, called end-organs, and finally the ganglionic or sympathetic nervous system is included in the peripheral system.

The Arrangement of the Nervous System.—The subdivisions of these parts, and their descriptions in detail, belong to general anatomy. But there have been so many special subdivisions, and particular names given to them in recent years, that I deem it necessary, in order to prevent confusion, to describe briefly the subdivision accepted by modern anatomists. The names here used are those adopted by the committee on anatomical nomenclature of the German Anatomical Society, and they have also been adopted by a large number of writers on neuro-anatomy.

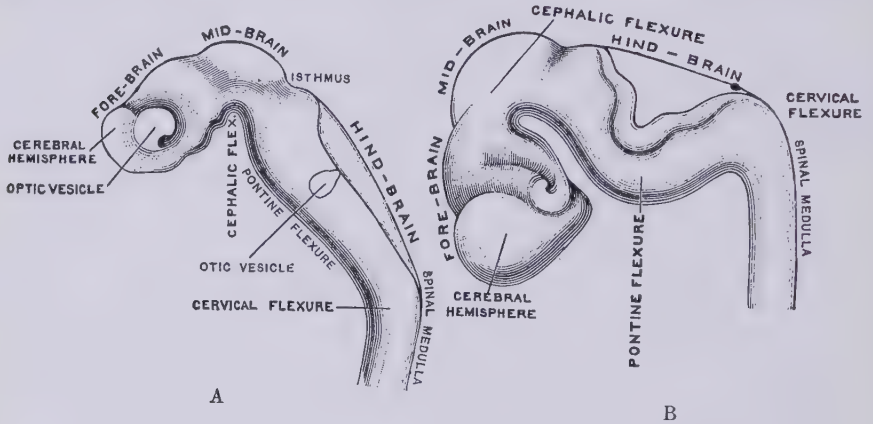


FIG. 1.—Showing divisions of embryonal human brain in third and fifth weeks. (*His.*) (*Cunningham.*)

Beginning with the brain, we find that its particular subdivisions are based upon the embryological development of this organ. As will be shown in more detail later, the brain is developed out of three vesicles, known as the *anterior*, *middle*, and *posterior* vesicles (Fig. 1). The most anterior of these vesicles is the *prosencephalon* or *anterior brain;* the middle vesicle becomes the *mesencephalon* or *mid-brain*, and the posterior vesicle develops into the *rhombencephalon* or *posterior brain.*

The anterior vesicle develops two secondary vesicles: the anterior portion of these, including the corpora striata, olfactory lobes and the cerebral hemispheres, forms the *telencephalon* (Fig. 2, I^2), while the hinder portion of this vesicle, which includes the thalamus and mammary bodies, forms the *diencephalon* (I^1). The middle vesicle is the *mesencephalon*, and it includes the corpora quadrigemina and cerebral peduncles (II).

The posterior vesicle is divided, from before backward, into three different parts: (1) the isthmus, which includes the superior cerebellar peduncles and valve of Vieussens, and part of the cerebral peduncles; (2) the *metencephalon* or *hind-brain*, which includes the cerebrum and pons Varolii; and (3) the *myelencephalon* or *after-brain*, which includes the medulla oblongata.

Brain.	Prosencephalon (anterior brain). I^1 and I^2	1. Telencephalon.	Hemispheres. Pars optica hypothalami.
		2. Diencephalon.	Pars mammallaris. Thalamus.
	Mesencephalon (middle brain). II	3. Mesencephalon.	Pedunculi cerebri. Corpora quadrigemina.
	Rhombencephalon (posterior brain). III and IV	4. Isthmus.	
		5. Metencephalon.	Cerebellum. Pons.
		6. Myelencephalon.	Medulla oblongata.

These different parts can be understood better by means of the accompanying figure (Fig. 2), which represents in a schematic way the brain of a mammal.

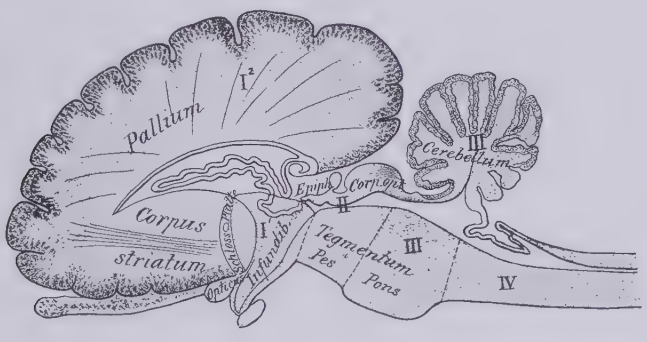


FIG. 2.

They are intimately connected by strands of nerve-fibres, and are connected closely also with the next portion of the nervous system, the spinal cord. The brain and spinal cord are spoken of as the cerebrospinal axis, and this is in close relation with the peripheral nervous system.

This peripheral nervous system is composed of two portions—first, the cerebrospinal mixed nerves, whose origin, distribution, and relations are comparatively easy to follow; and second, the autonomic or sympathetic nervous system. This portion of the nervous system is composed of two sets of ganglia—one, the vertebral ganglia, *i.e.*, the chain of ganglionic masses on each side of the vertebral column, and of certain ganglia connected with the cranial nerves; secondly, a very large number of

ganglionic masses distributed in the viscera, and known as the peripheral ganglia. The sympathetic nervous system is made up of multipolar cells and of some medullated (white), but mostly of non-medullated (gray) naked nerve-fibres.

Efferent white fibres pass out from the spinal cord through the anterior roots and pass partly to the sympathetic ganglia and partly continue on to the viscera, glands and blood-vessels. Afferent fibres originate in the sympathetic ganglia and pass in part through the posterior roots to the spinal cord; in part they turn and pass with peripheral nerves to the skin, blood-vessels and glands.

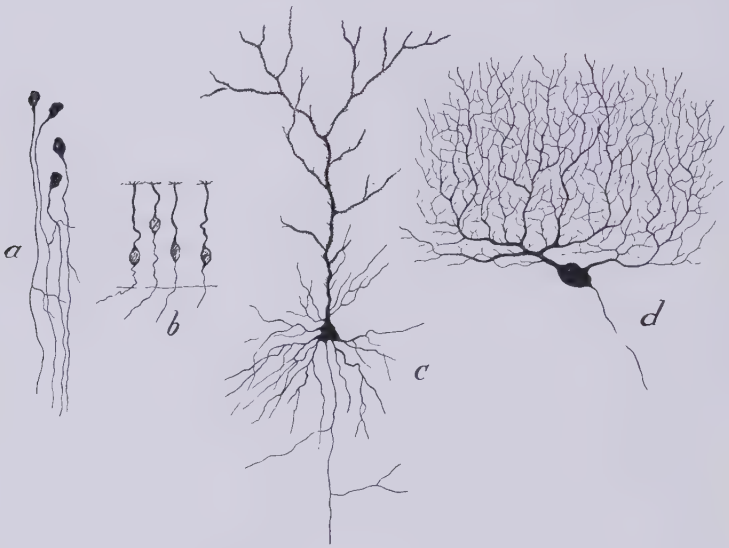


FIG. 3.—Nerve cells of different types. *a*, Unipolar; *b*, bipolar; *c*, pyramidal cell; *d*, Purkinje cell. (*Villiger.*)

THE GENERAL HISTOLOGY OF THE NERVOUS SYSTEM

The nerve-cells form the central body of the neuron and are minute objects varying much in size. The largest are 0.1mm. ($\frac{1}{250}$ in.) in diameter and are almost visible to the naked eye. The smallest are 0.7μ ($\frac{1}{3500}$ in.) in diameter; so that the average diameter is rather greater than that of a white blood-cell. In shape nerve-cells are for the most part irregularly spheroidal, but some are pyramidal, others spindle- or flask-shaped, and others globular. They all give off one or more fine processes or poles and hence, in accordance with the number of these, the nerve-cells are often spoken of as multipolar, bipolar, or unipolar.

In most cells one of the processes is continued on a long way and finally becomes a nerve-fibre. This process is called the axis-cylinder, or

neuraxon or *axon* (Figs. 3, 4 and 5 and Plate II). The other processes are relatively short and are called protoplasmic processes, or *dendrites*. The nerve-cell, then, is a protoplasmic body giving off several dendrites and usually a single neuraxon, the whole forming the neuron. The dendrites branch off irregularly and subdivide, but never anastomose. In some parts of the nervous system they have upon them little nodules or buds, and in the cerebral and cerebellar cortex these are so numerous as to give

Fig. 4.

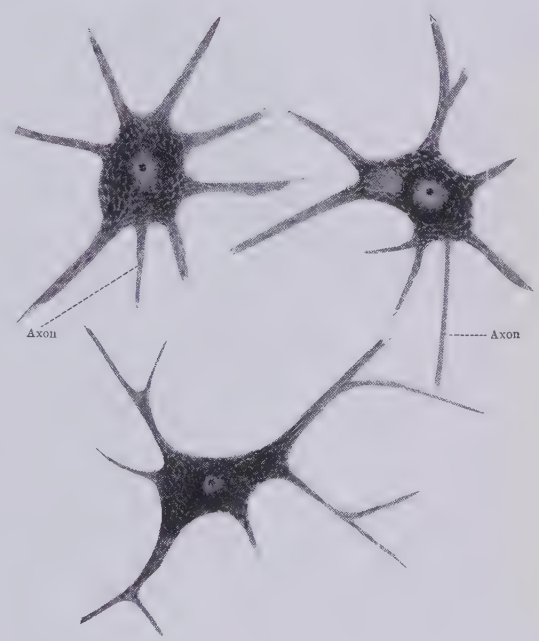


Fig. 5.

Fig. 4.—Nerve cell showing dendrites, axon, collaterals. *a*, Axis-cylinder-bifurcating at *b; c*, a collateral; *d*, varicosities of the dendrites. (*Cajal.*)

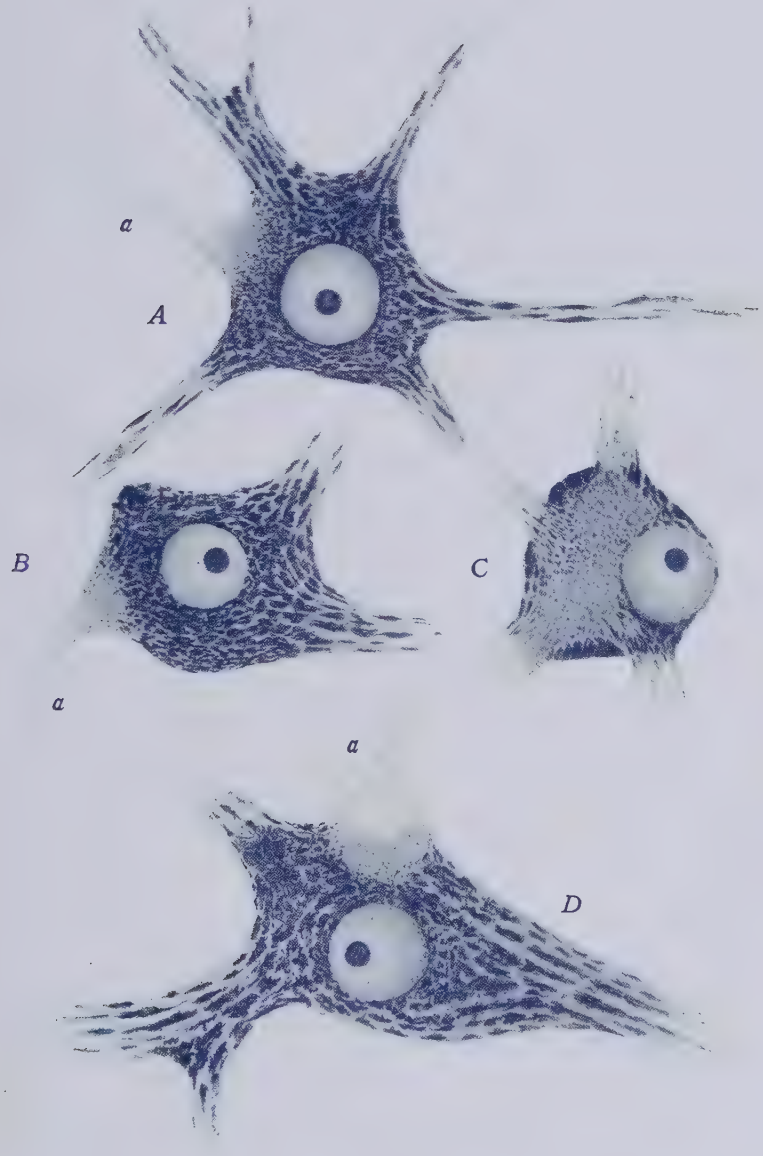
Fig. 5.—Nerve cells from anterior horn of spinal cord. (*Cunningham.*)

them the appearance of budded stalks. The dendrites are usually not very long, but in some cells they extend a very great way, reaching many times the diameter of the cell. The axis-cylinder process, or neuraxon, is given off directly from the body of the cell, as a rule (Fig. 5). It very soon becomes clothed with a thin sheath (myelin sheath), and as it passes along gives off branches at right angles, which form what are known as the *collaterals*. The neuraxon and collaterals finally end by splitting up into a

number of fine branches, which lose their myelin sheath and form the *end-brush* or *terminal arborization*. In some neurons instead of an end-brush, there are several minute ovoid bodies which lie upon the cell or its dendrites. They are called "end-buds" or terminal "buttons." They are seen especially in the medulla and pons, but not in the cerebral or cerebellar cortex. The axis-cylinder process or neuraxon does not anastomose with other cells either through its own end-brush or through the end-brushes of its collaterals. The end-brushes, however, pass in among the dendrites of other cells, and sometimes closely surround the cell-body. In this way one neuron comes into very intimate relation with others, but there is never any true union. *Each neuron of the nervous system is an independent unit.*

FIG. 6.—Ganglionic nerve cells, ultra-microscopical illumination. (*Marinesco.*)

The cell-body proper is composed of a *cytoplasm*. Within this lies the nucleus and within the nucleus a nucleolus. The body proper is not homogeneous, but is made up of a network of fine fibres or fibrillæ which pass in bundles from dendrite to dendrite and from dendrite to the neuraxon, called the endocellular fibrillar network. Within its meshes and arranged in a rather definite manner are certain stainable bodies called Nissl or chromophilic granules (Plate II). The chromophilic granules are arranged differently in cells of different function. They are believed to represent the functioning substance of the cell, while the fibrillæ form a conducting part. The cell-body usually contains a little pigment.



NERVE-CELLS STAINED BY NISSL'S METHOD, WITH TOLUIDIN BLUE. Magnified 675 diameters. (*Schaefer.*)

A, From anterior horn of spinal cord, monkey; *B* and *C*, from facial nucleus, dog; *D*, from reticular formation of pons Varolii, dog; *C*, shows Nissl degeneration, consequent on section of the facial nerve 15 days previously; *a*,*a*, axons.

The nucleus of the cell is a comparatively large spherical body, which is also made up of a reticulated structure known as the *chromatin network*. The chromatin consists of granules resting on a network formed by the achromatic substance (linin). The chromatin is practically identical with nuclein. Within the nucleus is a smaller body, known as the nucleolus, which stains still more intensely.

Nerve-cells are surrounded by a pericellular space, but are not inclosed in capsules, excepting those of the posterior spinal and vertebral ganglia. The reason for this is that within the central nervous system there is no neurilemma (nucleated sheath of Schwann); such sheath being found in the peripheral system only.

Marinesco has shown by studying the anatomy of the fresh nerve-cell by means of the ultra microscope some new details of the structure

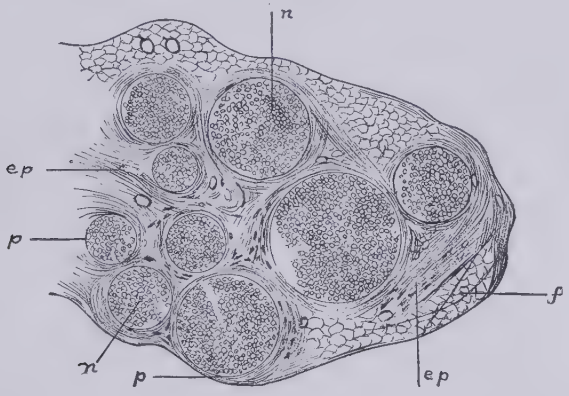


FIG. 7.—From a transverse section through the sciatic nerve. *ep*, Epineurium; *p*, perineurium; *n*, nerve fibres constituting a nerve bundle of fasciculus in cross-section; *f*, fat tissue surrounding the nerve. (*Klein.*)

of the cell. The body of the cell is composed of a complex of colloidal substances whose particles are illuminated by the lateral light of the ultra microscope. Fig. 6 shows four cells taken from the ganglion of a dog, examined in the serum of the same animal.

The nerve-cells of the sympathetic or vertebral ganglia are very like the central nerve-cells in the anterior horns. They are multipolar in shape and have dendrites and a neuraxon. The neuraxon goes to other neighboring cells, or it passes on to the spinal cord or the periphery. The cell is surrounded by a connective-tissue capsule lined with a flat epithelium, like those of the posterior spinal ganglia. The peripheral or terminal sympathetic nerve-cells lying in the viscera resemble those of the vertebral ganglia.

Nerve-cells are classified in accordance with their shape and number of processes. The multipolar cell is the common type and is found

throughout the brain, cord, and sympathetic ganglia. Bipolar cells are found chiefly in the column of Clark of the spinal cord and in spinal ganglia. Small nuclear cells and flask-shaped or Purkinje's cells are found in the cerebellum. Besides these there are described in the brain cortex angular, granular, pyramidal, globose, and spindle-cells.

The nerve-fibres of the nerve-centres are found chiefly in the white tissue or white matter. In the periphery they form the nerve proper of gross anatomy. The peripheral nerve is composed of bundles of nerve-fibres called nerve fasciculi (Fig. 7). It is surrounded by a connective-tissue sheath called the sheath of Henle, or epineurium. From this sheath, connective-tissue fibres pass in and surround the fasciculi. The sheath of the fasciculus is called the perineurium. From the perineurium, strands of connective tissue run in among the ultimate nerve-fibres, forming the endoneurium (Figs. 8 and 9). Lymphatic spaces lined with endothelium exist in the layers of the peri- and endo-sheaths. In the nerve-centres, the nerve-fibres, have no such sheaths, but are supported by a connective tissue and neuroglia framework.

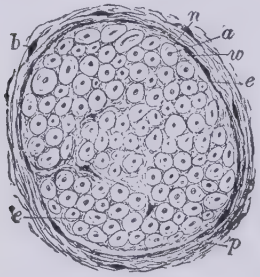


FIG. 8.—A simple funiculus more highly magnified. The apparent small nucleated cells are sections of the nerve-fibres and their axis-cylinders. *a*, Axis-cylinder, *w*, white substance of Schwann or medullary substance; *n*, neurilemma; *e*, endoneurium; *p*, perineurium; *x*, connective-tissue cells of the same. (*Piersol.*)

The nerve-fibre is a long fine strand of tissue varying in diameter. It may be white or gray, according to whether it has or has not a myelin sheath. It is composed from within out of (1) an axis-cylinder, (2) a myelin sheath, and (3) a neurilemma. (1) The *axis-cylinder* is the essential part of the nerve. It is the prolongation of the neuraxon of a nerve-cell and consists of protoplasm. It is itself made up of fine fibrillæ (primitive fibrillæ) which run longitudinally.

The myelin sheath is developed, like the axis-cylinder, from the ectoderm, and is closely related nutritionally to the axis-cylinder, which it protects and isolates. (3) The *neurilemma* or *nucleated sheath* is a delicate covering forming the outermost sheath of the nerve. It is also of ectodermic origin. The sheath is absent in the fibres of the central nervous system and in some fibres of the periphery.

Variations in the Types of Fibres.—In accordance with the arrangement of the sheaths of the nerve-fibres, several kinds are described. The principal types are the *medullated* and *non-medullated.*

Medullated nerve-fibres make up the bulk of the white matter of the brain and cord and cerebrospinal nerves. They consist of a myelin sheath and axis-cylinder, and may or may not have a neurilemma.

Fibres with myelin sheath, but without a neurilemma, make up the white matter of the central nervous system.

Non-medullated fibres, or fibres of Remak, occur principally in the sympathetic system, but they are also found in the cerebrospinal nerves. They are grayish and faintly striated, and consist of axis-cylinders, with a thin, homogeneous, nucleated sheath, the neurilemma, lying directly upon them.

Naked axis-cylinders are found in the peripheral terminations of nerves as well as in the brain and cord and sympathetic.

Size.—The nerve fibres are of two kinds as regards size. The small fibres are about 2μ or $\frac{1}{12000}$ inch in diameter, the large 20μ or $\frac{1}{1200}$ inch. The small fibres are connected with smaller cells, and either run a shorter course or are distributed to the involuntary muscular

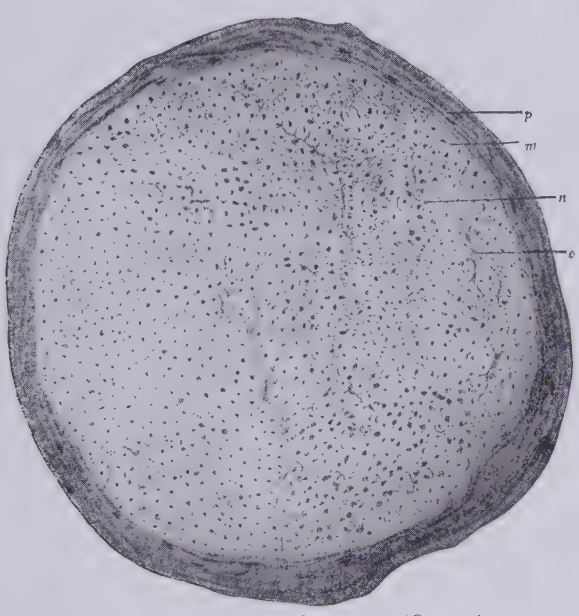


FIG. 9.—Section of nerve. (*Quain.*)

fibres of the blood-vessels and viscera. The motor fibres are larger than the sensory.

The white fibres of the sympathetic nervous system are about one-third smaller than the ordinary cerebrospinal or somatic fibres.

The peripheral nerve-fibres, except the optic, have no neuroglia; they terminate in fine fibrillæ among epithelial cells, or in special end-organs.

The *central nervous fibres* make up the white matter of the brain and cord. They are, like the peripheral nerves, the prolongations of the

neuraxons. They are composed of an axis-cylinder process and myelin sheath, but have no neurilemma, and probably no nodes. At frequent intervals each fibre gives off branches at right angles forming the "collaterals."

Connections of Nerve-cells and Nerve-fibres.—One nerve-cell is never connected directly with another, so far as anatomical investigation can show. One nerve process becomes an axis-cylinder, receives a myelin sheath, gives off collaterals, and finally breaks up into a fibrillary "end-brush" surrounding a cell, but not passing into it. There is physiological, but no apparent anatomical continuity.

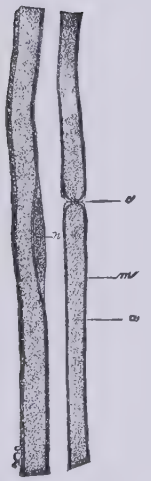


Fig. 10.

Fig. 11.

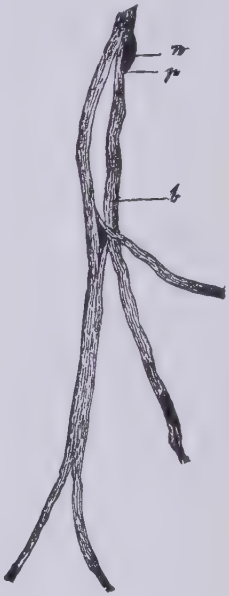


Fig. 12.

Fig. 10.—Medullated nerve fibre. *a*, Axis cylinder; *n*, nucleus; *m*, medullary sheath; *c*, node of Ranvier.

Fig. 11.—Medullated nerve fibre, showing mode of division.

Fig. 12.—Non-medullated nerve fibre. *n*, Nucleus; *b*, striations.

The Neuroglia.—The supporting tissue of the peripheral nerves is connective tissue only; that of the central nervous system is connective tissue and, in addition, a peculiar substance called neuroglia. The neuroglia or supporting tissue of the nervous centres is derived from the ectoderm. It is composed of cells with very numerous and finely ramified processes, which make a supporting network about the nerve-cells and fibres (Fig. 13). The cell-body is composed of granular protoplasm, lying in which is a large nucleus, within which is the nucleolus. The body of the cell is small in amount in proportion to the nucleus. The fibrillary processes form a felt-like network, and

in regions where there is much neuroglia tissue this looks like a homogeneous matrix. It is, however, made up of the fine fibrils.

The Non-nervous Tissue—*The Blood-vessels.*—The peripheral nerves are richly supplied with blood. Each nerve receives arterial supply

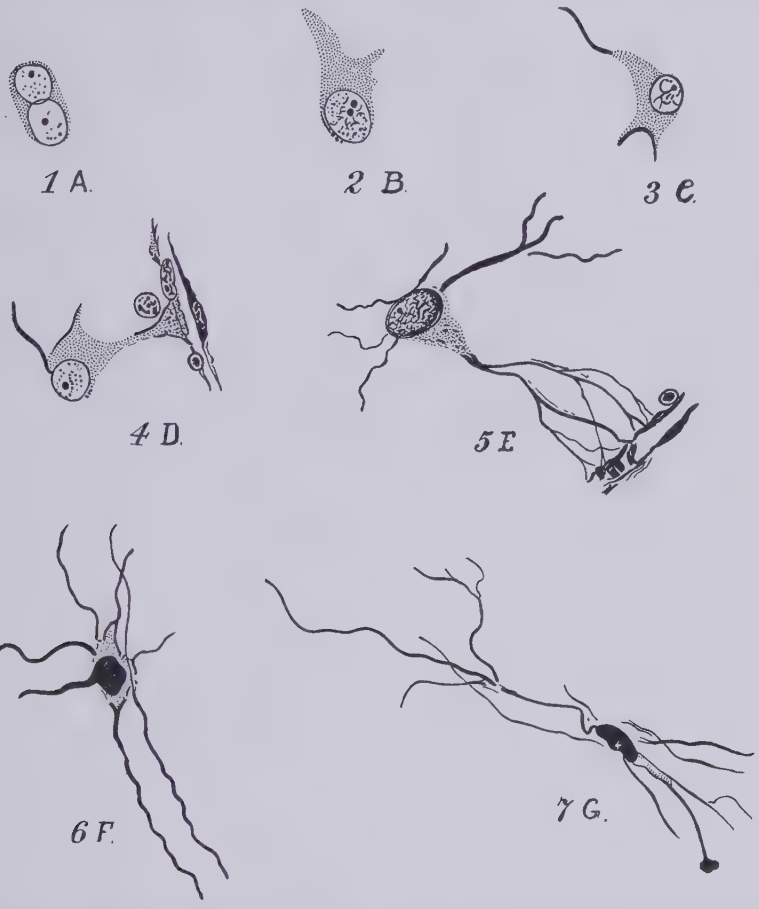


FIG. 13.—Phases in the development of neuroglia cells and fibrils. 1*A*, Dividing neuroglia nuclei surrounded by protoplasm; 2*B*, protoplasmic processes more definitely formed; 3*C*, commencing condensation of protoplasmic processes producing darkly staining fibril; 4*D* and 5*E*, mode of attachment of the processes to a vessel wall and differentiation of foot into fibrils; 6*F*, further development of fibrils; 7*G*, protoplasm almost entirely differentiated into fibrils and nucleus shrunken. (*From figures drawn by Dr. George A. Watson.*)

from many different branches, but always from the same general source. The artery passes to the nerve-sheath obliquely, then divides dichotomously and sends branches a long distance up and down on the sheath. It may pierce the sheath, however, first, and then divide, as above de-

scribed. The dichotomous branches send off arterioles and capillaries, which form plexuses about the nerve fascicles. These are "the interfascicular arcades." The arteries subdivide in such a way as to prevent sudden impact of a large blood-stream into the tissue of the nerve. In this respect the nerve circulation resembles that of the brain and cord. The veins subdivide dichotomously, like the arteries. They freely anastomose with the muscular veins, so that muscular action helps nerve circulation. The veins of the superficial nerves connect with those of the deep nerves.

The blood-vessels of the spinal cord and brain will be described later.

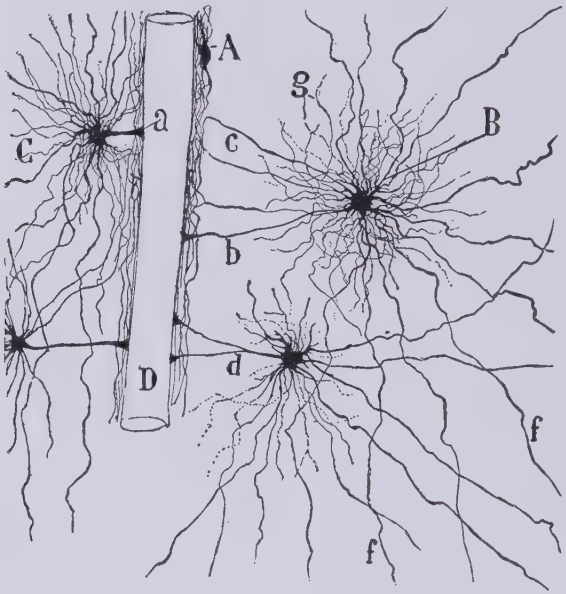


Fig. 14.—Glia cells as impregnated in silver-chromate preparations. (*After Ramón Cajal.*)

Lymphatic vessels and spaces are found in the epineurium and perineurium. There are no distinct lymphatics in the fasciculi, but lymph-spaces probably exist.

THE NEURONIC ARCHITECTURE OF THE NERVOUS SYSTEM

Having described the component parts, I shall now show the way in which these parts are arranged to form the nervous system.

The nervous system, as already shown, is composed of single nerve units that are called *neurons*, and the neuron is made up of a cell-body and numerous processes, one of which is the neuraxon, the others the dendrites.

The neuraxon has always the function of carrying impulses away from the cell; it is a cellufugal fibre; the dendrites, however, bring impulses to the cell, and are cellupetal in function. The dendrites are in contact with end-brushes or end-buds of the neuraxons of other cells, and in this way receive the nerve impulse and transmit it to the cell-body. The nervous system is thus a mass of neurons which are packed closely together, and form with each other most intricate relations, but never connect directly one with another. No nerve-fibre or dendrite of one cell anastomoses with that of another, as blood-vessels do. Each neuron is anatomically independent.

It is the purpose of this neuronic mass to receive impulses from within or without the body, to transfer and modify them, and to send out impulses in such way as properly to control the vital functions and keep the individual in proper harmony with his environment. The nervous system is a great receiving, regulating, controlling, and discharging machine, the machinery being the neurons, the force that works in it being called nervous energy. The neurons as a whole have this force stimulated or aroused in them. The nerve-cell co-ordinates and distributes it. The nerve-cells are massed together for the most part in the brain and spinal cord, forming the gray matter, while the neuraxons as distributors make up the white matter and the cranial, spinal, and sympathetic nerves. It is convenient to make a division, therefore, into the central or *somatic* nervous system, with its peripheral nerves, cranial and spinal, and the *sympathetic* or *autonomous* nervous system.

The Somatic Nervous System.—Since the nerve-fibres of the brain and cord are white in texture, while the cells in mass are of gray color, it is very easy to distinguish the deposits of cells from the fibres and thus make subdivisions of the central nervous tissue. One portion of this gray matter is found deposited in the centre of the spinal cord, extending up to the floor of the medulla, thence underneath and around the aqueduct of Sylvius to the floor of the third ventricle. This is called the *central gray matter*. Another deposit, much larger in amount, covers the whole of the cerebrum and cerebellum, and forms the cerebral and cerebellar *cortex*. Smaller deposits make up the great *basal ganglia*, corpus striatum, optic thalamus, and corpora quadrigemina, besides several small deposits (the *small basal ganglia* cerebral and cerebellar), such as Luys' body, the red nucleus and Deiter's nucleus.

The peripheral nervous system contains nerve-cells, as well as fibres. Their anatomical arrangement is easily understood, but their relation to the central nervous system is less simple.

The sympathetic nervous system consists of (*A*) an autonomic or parasympathetic portion and (*B*) a sympathetic system proper (Fig. 15).

The autonomic nervous system is in general distinguished from

the sympathetic proper by the fact that it does not at first pass into any ganglion, but runs in cranial or spinal nerves until it reaches the periphery. It then enters a ganglionic mass, and from this it sends fibres to the peripheral organ.

(*A*) This autonomic system consists of a mid-brain, a hind-brain

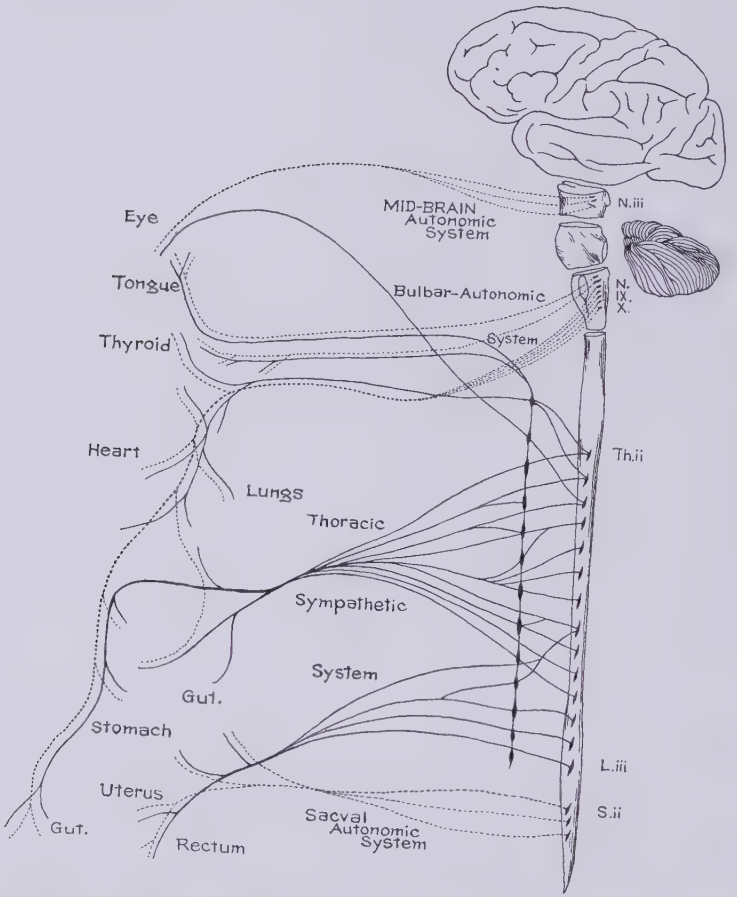


FIG. 15.—Scheme of the autonomic and sympathetic system proper, according to Langley.

and a sacral portion. The mid-brain autonomic system sends out fibres which run into the third nerve to the ciliary ganglion and thence fibres to the ciliary muscles and the sphincter of the iris.

The hind-brain autonomic system sends fibres which run into the nerve of Wrisburg and the seventh nerve; the glossopharyngeal and the vagus. The fibres that run in the nerve of Wrisburg and the seventh

nerve go to the submaxillary and the sublingual ganglia and thence supply the vasodilator nerves to the tongue, submaxillary and sublingual glands and secretory fibres and vasomotor fibres to the mucous membrane of the nose, soft palate and upper pharynx.

It sends fibres via the glossopharyngeal nerve to the otic ganglion and these control secretion and vasodilation in the parotid gland.

The most important parts of the hind-brain autonomic fibres run in the vagus nerve. They go to the jugular ganglion and the ganglion of the trunk and of the vagus and thence send motor and secretory fibres to the œsophagus, stomach and small intestines, as far as the ileo-cecal valve.

The sacral autonomic system consists of fibres which pass out through the second and third sacral nerves and are connected with the ganglia of the hypogastric plexus. They send fibres which supply motor fibres to the bladder and colon and rectum, inhibitory fibres to the sphincter of the bladder and rectum.

In a general way it is found that the autonomic and sympathetic systems antagonize each other in their action, thus the fibres of the autonomic system contract the pupil; those of the sympathetic dilate; the fibres of the hindbrain of the autonomic system inhibit the heart and stimulate movement and secretion, while the sympathetic fibres antagonize this action. The same is true though to a less marked extent of the sacral portion of the autonomic system.

	Autonomic or Para-sympathetic.	Sympathetic.
Mid-brain portion.	Contracts pupil.	Dilates pupil.
Bulbar portion.	Coronary blood-vessels.	Coronary blood vessels.
	Inhibits heart-action.	Accelerates heart-action.
	Dilates blood-vessels.	Contracts blood-vessels.
	Inhibits sweat-glands.	Stimulates sweat-glands.
	Contracts muscle-walls of œsophagus, cardiac sphincter, stomach,	Relaxes muscle-walls of œsophagus, stomach, cardiac sphincter,
	Stimulates gastric-secretion.	
Sacral portion.	Dilates blood-vessels of rectum, anus. external genitals.	Contracts blood-vessels of rectum, anus. external genitals.
	Contracts muscles of colon, rectum, anus, external genitals, bladder, urethra.	Relaxes same

(*B*) The sympathetic system proper consists of the two chains of vertebral ganglia, of their peripheral ganglia (cœliac, etc.), and of connecting fibres.

Both portions of the sympathetic system have efferent and afferent fibres. The efferent nerves never pass directly to the tissue to be innervated, but first go to ganglia, from which a second neuron sends out its fibres to the terminal organ or gland.

The afferent or sensory fibres are very few compared with the efferent, so that the viscera have no sensibility except on great and peculiar irritation. The sensations evoked reach the cord but the brain and consciousness are reached through the somatic sensory fibres which in general refer the pain to the corresponding somatic segments.

The sympathetic fibres to the viscera run mostly as independent nerves, but the fibres to the trunk and extremities and head run mostly in the trunks of the somatic nerves.

I come now to a description of the general arrangement of these various nerve units; and here I must suppose that my reader has a knowledge of the ordinary anatomy of the subject.

The nerve-cells of the ganglia on the posterior spinal roots furnish the best starting-point in an attempt to trace out the connections. These cells give off a single process, which quickly divides in a T shape. One branch of the T passes peripherally through a mixed spinal nerve to the skin, forming a sensory nerve. The other passes centrally, enters the posterior spinal roots, and breaks up into little filaments, which surround a nerve-cell in the posterior horn or analogous nuclei. This forms the first or outer sensory neuron. The outer branch of the spinal ganglion cell which went to the periphery as a sensory nerve was its *dendrite*, or protoplasmic process, which has evolved into a sensory nerve and is *cellupetal* in function. The other process is the neuraxon proper and it is *cellufugal*, carrying impulses away from the ganglion cell into the cord.

The next neuron begins as a cell in the posterior horn, or in like parts. It sends a neuraxon up the spinal cord, a collateral branch passes to the cerebellar cortex, while the direct fibre surrounds a cell in the optic thalamus. This forms the second sensory neuron. The cell in the thalamus gives off a neuraxon which passes to the gray matter of the cerebral cortex, and here it either directly affects the cells in this region or does it through the medium of another shorter neuron, which is called "associative."

Thus each sensory impulse from the periphery reaches the conscious centres of the brain by passing along three or four neurons. The primary neuron in all cases lies mainly outside the central nervous system and forms a sensory nerve. The sensory nerves do not there-

fore *arise* in the cord or medulla, but have their terminal nuclei there. Afferent nerves serve the purpose of conducting impulses which arouse sensation, and reflex action.

The sensory nerves of the surface of the body are called *extero-ceptive* when they excite reflex action. They are more closely associated with the cerebrum and the activity of the skeletal muscles. The visceral nerves which excite reflexes are called *proprio-ceptive*. They are associated with the cerebellum, and the visceral muscles. They give rise

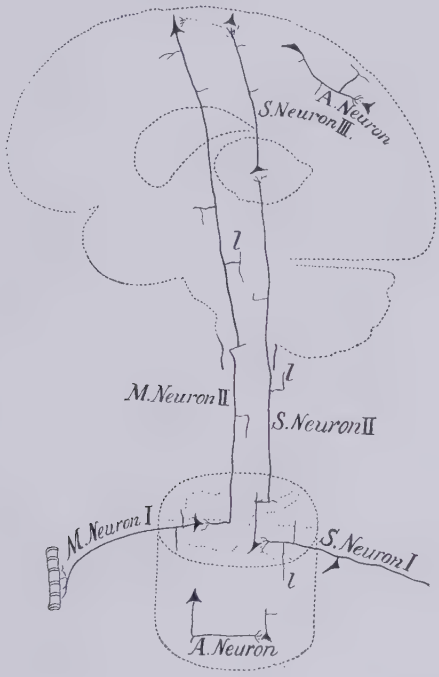


FIG. 16.—Diagram showing the arrangement of the neurons or nerve units in the architecture of the nervous system. *M. Neurons I.* and *II.*, Motor neurons; *S. Neurons I., II., III.*, sensory neurons; *A. Neuron*, associative or commissural neuron.

to the tonic reflex which keeps the voluntary muscles at a certain degree of tension (Sherrington). (See also Fig. 18 p. 33.)

The neurons of the brain cortex cannot yet be distinctly classified, and I shall not attempt it here at all. The matter will be brought out more fully in connection with the anatomy of the brain. It is sufficient to say that nerve units connect together the cerebellum and cerebrum with the basal ganglia, the frontal lobes and the cerebellum, the two hemispheres of the cerebrum, and different areas of the cerebral cortex. Leaving out of consideration these neurons, which are largely psychic in function, we start with the large motor cells in the central

convolutions of the brain. These send down neuraxons, which pass into the spinal cord and surround the cells of the anterior horns. They form the upper or central motor neurons. The anterior horn-cells send off neuraxons, which pass out through the anterior roots and thence to the voluntary muscles. These are the lower or peripheral motor neurons. These motor neurons have a double function—that of exciting muscles to contract, and that of inhibiting muscular activity and causing them to relax. The *efferent nerves are almost equally inhibitory and motor*. With each voluntary muscular act impulses to contract, excite for example the flexor groups, and impulses to relax, cause the opposing extensors to relax. The motor mechanism is a motor-inhibitory mechanism. This has been especially established by Professor Sherrington. Besides this there are groups of cells in the lateral horns and central parts of the spinal cord which send off neuraxons that also pass through the anterior root, but they leave the cerebrospinal nerves and enter the vertebral sympathetic ganglia. Here they in part surround the cells of these ganglia and have their terminals there. These sympathetic ganglion cells in turn send neuraxons, which pass in the sympathetic nerves to the peripheral ganglia, where they meet a third group of neurons. They also connect with the other ganglia of their own class and send neuraxons through the posterior spinal roots to the cord. It is not known with what neurons higher up in the nerve-centres the lateral horn-cells are connected, but probably with cells in the thalamus.

Such in outline is the neuronic architecture of the nervous system. I do not attempt here to work out the neurons of the special senses, nor to introduce the spinal cerebellar neuron. This will be done later. It is sufficient to say that the studies in this direction show a marvellous harmony as well as beauty in nature's scheme.

The neuronic architecture is shown in the accompanying diagrams (Figs. 16 and 18).

GENERAL PHYSIOLOGY

The Peripheral Neurons.—The nerves which run between nerve-centres and end-organs carry impulses each way. They are, therefore, divided into the *afferent*, centripetal or ingoing, and *efferent*, centrifugal or outgoing. The old division into motor and sensory nerves will not answer, for there are many outgoing nerves which are not motor. The *afferent* nerves are:

1. The sensory, including:

- Nerves of general sensation.
  - Pain nerves or pathic nerves, heat and cold or thermic nerves.
- Nerves of special sensation.
  - Tactile, including
    - Contact,
    - Pressure,
    - Locality.
  - Nerves of muscular and articular sense.
  - Nerves of special sense of smell, sight, taste, hearing and space.

These carry
(*a*) Epicritic sensations and
(*b*) Protopathic sensations.

2. Excito-reflex nerves.
   - Extero-ceptive.
   - Propero-ceptive.

Protopathic and Epicritic Sensibility.—According to Dr. Henry Head, the afferent nervous system is composed of two different sets of sensory fibres, called the protopathic and the *epicritic.*

The *protopathic* set or system which is distributed to the skin and viscera, and to the muscles.

(*a*) The fibres to the skin run in the peripheral nerves and give to it a low degree of sensibility. The fibres to the viscera run in the sympathetic and give to it also a low degree of sensibility.

(*b*) A set of deep fibres to the muscles which supply the sense of deep pressure. These fibres run in the muscular nerves and are connected with the Paccinian bodies.

The "protopathic system," then, as a whole, consists of fibres of sensibility which supply the skin, the viscera, and all parts of the body, inside and out. It furnishes a low degree of sensibility to the viscera, muscles and to the skin. This system enables one to appreciate a sense of pain and temperature, though not to a very delicate extent.

There is another system of afferent fibres which supply the skin alone, a purely cutaneous system, and this is called *epicritic.* This epicritic sensibility enables us to appreciate light touch, the points of the compass, localization, and minor degrees of temperature, ranging between 22° and 40°C.

Epicritic sensation gives us the sense of the point (a variant of appreciation of relative size).

Protopathic sensation gives sense of pain apart from the knowledge of the pain-producing object.

The epicritic fibres furnish a delicate and localized appreciation of pain. Protopathic fibres carry sensations which are badly localized, widely diffused and sometimes referred to other parts than that of the stimulation. The protopathic fibres are incapable of appre-

ciating light touch, and minor degrees of heat and cold, and pain appreciation is a diffuse tingling and thrilling sensation. This distribution of sensory fibres exists only in the peripheral nerves. In the nerve-roots and central nervous system the systems are fused. [The above description of the views of Dr. Head and his associates has been seen and corrected by Dr. Head, of whose courtesy I make acknowledgment.]

The *efferent* nerves are:

1. Motor nerves, going to voluntary or striped muscles, heart muscle, smooth muscle, including the vasoconstrictor and dilator nerves.

2. The secretory. These act upon glands. Impulses to the blood-vessels (vasomotor) generally accompany the secretory impulses.

3. The inhibitory. These nerves control muscular movements, secretion, perhaps also nutrition.

Central Neurons.—There are intercentral or associative neurons, which connect different parts of the cerebrospinal system together. Some of these connect symmetrical parts on each side together and have a coordinating function. Others connect higher with lower centres and carry ascending and descending impulses.

End Organs.—In the peripheral nervous system we have *end-organs*. These are delicate and in some cases complex arrangements of nervous and other tissue at the periphery of the nerves. Their object is to allow the nerves to be irritated by special *stimuli* which would not otherwise affect them, *e.g.*, light or sound. Their object is also the proper utilization of efferent impulses upon other tissues. There are end-organs, therefore, for both sensory or afferent and for efferent nerves. The end-organs of the afferent nerves are—eye, ear, taste-buds, corpuscles in the Schneiderian membrane, various tactile cells and bodies, the space sense-organ, the muscle-spindles.

For the efferent nerves—neuro-muscular corpuscles in the voluntary muscles, local ganglia about the arteries, local ganglia in the glands. Trophic end-organs are not known. In many cases the end-organ is nothing but the terminal fibre of the nerve. This loses both medullary sheath and neurilemma, leaving only the axillary cylinder. It then splits up into a terminal plexus, or else without splitting passes between and around the cells which it is to affect.

The Work of the Cell-body of the Neuron.—In the working of these mechanisms the nerve-cell body is the agent which in some instances generates, but more generally distributes and directs the impulses which pass to it. The larger the nucleus of the cell in proportion to its protoplasmic body, the more stable or less sensitive the cell. The larger the amount of protoplasm relative to the nucleus, the more active the discharging power of the cell. The nucleus is the part of the cell-body which

is essential to constructive metabolism. By means of it the cell builds up its protoplasmic substance. When the nucleus dies, the cell may live or function for a time, but it lives only on what has been stored up; it can build no more and soon dies. Nerve-cells with few exceptions (spinal ganglia) have no centrosomes; they cannot divide and multiply. Once dead they cannot be restored.

The nerve-fibres conduct impulses variously generated. These impulses travel at the rate of about 100 to 120 feet per second. It is less in visceral nerves (25 to 30 feet per second). Nerve-cells are exhausted by successive stimulations, but nerve-fibres can be continually excited and are practically non-fatiguable. There are no electrical currents in normal living nerves (Landois) except when an impulse travels along them. Then an electrical current travels along with the impulse. It is called the current of negative variation. The irritability or excitability of a nerve is the power it has of responding to a stimulus. When a constant electrical current is passed along a nerve its irritability is modified. This modified condition is called *electrotonus*. When a nerve-impulse passes up an afferent nerve and is then reflected along an efferent nerve, it is called a *reflex action*. The time required for this process is called the *reaction time*. This averages from 0.125 to 0.2 of a second.

CHEMISTRY

The specific gravity of nervous tissue is about 1.036; that of the brain is 1.038; of the spinal cord and nerves, 1.034 (Bischoff, Krause). The reaction is alkaline, but this is lessened by activity, owing to the development chiefly of lactic acid. The gray matter is less alkaline than the white.

The nervous system has the following composition (Baumstark, quoted by Hammarsten):

	White Matter	Gray Matter
Water in 1,000 parts	695.35	769.97
Solids	304.65	230.03
Protagon { Cerebrin / Lecethin (neurin) }	25.11	10.08
Insoluble albumin and connective tissue	50.02	60.79
Cholesterin	45.12	23.81
Nuclein	2.94	1.99
Neurokeratin	18.93	10.43
Inorganic salts	5.23	5.62

Water makes up nearly three-fourths of nervous tissue, there being more in the gray than in the white matter and least in the sympathetic nerves. The inorganic salts amount to about 0.5 per cent. The largest single constituent is phosphorus (Breed) combined with potassium,

sodium, magnesium, calcium, and iron, forming phosphate salts. Of the other constituents chlorid of potassium is the most important.

Protagon is a very complex substance of a fatty character, containing nitrogen and united with glycerin-phosphoric acid instead of glycerin. It is said by some to be made up of two bodies, cerebrin and lecethin, the latter containing an ammonia compound called neurin. Protagon is especially found in the white matter. The gray matter and axis-cylinders contain globulins, nucleo-proteid and nuclein, a very important substance in cell metabolism. Nuclein ($C_{29}H_{49}N_9P_3O_{22}$, Miescher) is composed of nucleic acid, a substance rich in phosphorus and a variable amount of albumin. The gray matter, *i.e.*, the nerve-cells, contains also various albuminous substances. Nucleo-keratin is found in the neuroglia and medullary sheaths. The nuclein and the allied substance nucleo-albumin are called albuminoids (Halliburton). They both contain phosphorus and are found chiefly in the nucleus. The albuminous substances, called also proteids by Halliburton, have little or no phosphorus, and make up the most of the cell-body or cytoplasm.

CHAPTER II

THE CAUSES OF NERVOUS DISEASES

Nervous diseases are produced in part by predisposing influences which may be likened to a fecund soil; in part they are due to exciting causes, which are like the seeds dropped upon the soil in the accidents of life.

Heredity is the most serious and important of these predisposing causes, in particular of those neuroses that are constitutional and are not the results of bodily accidents. A nervous disease, however, is rarely directly inherited. Parents do not pass down special maladies, but only a general tendency to nerve disease, which is not developed into any distinct trouble unless some disturbing cause arises. Nervous parents may have children who have unstable, over-irritable, and inadequate nervous systems. Such persons have what is called a *neuropathic constitution* or diathesis. This diathesis may be transmitted when the parents, though not especially neurotic, suffer from syphilis, alcoholism, and diseases of malnutrition, like tuberculosis. So far as the office of parentage goes, persons of great talent in affairs, or great artistic genius in any direction, may be counted as neurotic and are very likely to have children of neuropathic constitution. This is less apt to be the case when one parent is of stable and lymphatic type. If two persons having not simply a nervous constitution but distinct nervous or mental disease marry, their children are liable to serious nervous or mental disease. The intermarriage of blood relations, such as first cousins, does not lead to neurotic children, if the parents are not neurotic themselves, and are of robust health and dissimilar temperaments. Injuries or even severe shock to the mother during the early months of pregnancy sometimes leads to nervousness in the offspring. The mother transmits neuroses more often than the father. There are certain rare nervous diseases which appear in different branches and members of a family, such as an uncle, cousin, nephew, and son. These diseases may pass also by direct inheritance from parent to child, or may skip a generation. They are called "family diseases," and are of the nature of congenital defects, like webbed fingers or club-foot.

Degeneration is the name given to a condition in which there is a morbid deviation from the normal average in mental traits, physiological powers, or physical structure. It is a term most often used in connection with mental characteristics. It is most often an in-

herited state, and the word degenerate is often used to indicate a person who has a hereditary neuropathic or psychopathic constitution. Degeneracy in a moderate degree often accompanies great mental powers, especially of the artistic kind, and it is almost invariably associated with genius. It is quite compatible with mental soundness and a fair degree of physical health. It is then well to use the term, suggested by Walton, of *deviation*. Those who have unusual mental gifts and degenerate characteristics are called superior degenerates. The criminal and the insane and erratic and eccentric persons of weak judgment have also the neurotic constitution, and are called inferior degenerates. The weak-minded, imbecile, and idiots form the lowest class of degenerates, and are called the *debiles*. Degeneracy of constitution whether inherited or acquired is a condition which fits the person for acquiring nervous disease.

Age.—In infancy and early childhood, nervous diseases are rather frequent on account of the accidents at birth, the liability to infectious fevers, and malnutrition, and the high degree of sensitiveness of the yet immature nervous system. Still, a carefully watched infant is relatively safe. Motor disorders, such as paralyses, convulsions, and chorea, are much the more common troubles. At the time of puberty sensory disorders, such as headache, and migraine appear, and often epilepsy, hysteria, and disorders of sleep. Hereditary tendencies to nervous disease also begin to develop at this time or a little later. At the period of adolescence, the maladies already mentioned also may be brought out; but in addition neurasthenic, morbid sexual, hypochondriacal, and insane tendencies are seen. From maturity to the time when degenerative changes begin, forty to forty-five, the individual suffers from those nervous disorders brought on by accidents, injuries, prostrating attacks of sickness, over-strain, infections, indulgence in alcohol and narcotics, and the abuse of the bodily functions. At and after the climacteric, one sees oftenest such maladies as result from vascular disease, apoplexies, brain softening, also severe forms of neuralgia, and spasm.

Sex.—Sensory and functional disorders are more frequent in women; motor and organic disorders more frequent in men.

Condition and Occupation.—No general facts will be laid down here. Celibates, however, it may be said, suffer more from nervous disorders than married people. It will be shown later that certain occupations entail special nervous disorders and that indoor life promotes functional nervous diseases.

Work.—Hard, constant, excessive muscular work, leads to arterial sclerosis and the neuroses that result from a defective circulation.

Mental work produces no injurious effect upon metabolism and is a

healthful form of activity, even intense and long-continued mental work being comparatively harmless. It is the worry and strain and emotional excitement associated with hard work that does harm and leads to neurasthenic disorders.

Mental Attitude.—The expectant and untrained mind is much more liable to nervous disease as the result of injury, shock, etc. Little evil can befall a prepared mind. The Freudean school asserts that a person who has been "analyzed" is much less subject to the minor psychoses.

Habits.—Excessive indulgence in alcohol is a most prolific cause of nervous disease, chiefly by the action of this substance on the blood-vessels and the stomach. Excesses in eating, in tea-drinking, irregularity in sleeping, and bad habits of working predispose to nervous disease. Sexual excesses are usually the result rather than the cause of nervous disorders. They are the evidence of mental more than of nervous weakness. Bad mental habits acquired usually in early life as the result of wrongful education or a poor environment and example lead to many functional nervous disorders. By bad mental habits is meant especially the tendency to lay too much stress on trivial things, to worry and "fuss" over details, to have "precisions," to get "notions" and assume as guides in life foolish types of religion, philosophy or sanitation. The obsessive neurasthenic is one of the characteristic features of our American civilization.

Climate and Civilization.—Nervous diseases are most frequent in temperate climates, and in those which are dry and elevated. They increase with the progress of civilization and the greater strain, complexity, and luxury of modern social life. Those organic nervous diseases which are largely dependent on vascular disease are frequent in the poorer classes, among whom syphilis, alcoholism and bad feeding prevail. Functional and degenerative disorders are frequent in the higher classes. Nervous diseases, if we except those of the degenerative type, prevail more in urban populations.

Diathesis.—The rheumatic and gouty diatheses predispose to nervous troubles, more especially those which are of a peripheral and functional nature. Those allied conditions in which the products of tissue waste are not properly oxidized and eliminated, have a similar influence.

Trauma and Shock.—Exhausting hemorrhages and trauma may be the direct cause of or may predispose to nervous disease. Trauma and mental shock may cause functional diseases, such as neurasthenia, or may lead to the development of insanity or indirectly to degenerative organic disease. Mental shock, and especially a fright, oftener than severe bodily injury, leads to the development of functional neuroses. However, it is only the weak, the neurotic or unprepared who can be thus affected.

Infections.—In comparison with their frequency, the infective fevers are not great factors in producing nervous disease, but practically they often play an important part. Scarlet fever is the most dangerous disorder in this respect. Measles perhaps ranks next; then follow influenza, diphtheria, typhoid fever, and pertussis. Among chronic infections syphilis ranks first; tuberculosis, malaria, the pellagra, and beriberi are also to be mentioned. The importance of *syphilis* which causes perhaps one-half of organic nervous diseases will be dwelt upon later.

Poisons.—Tea, coffee, cocoa, tobacco and lead, mercury, copper, and arsenic, are to be placed among the causes of nervous disease. But methyl and ethyl alcohol are justly credited with exerting the most sinister influence on the nervous system of all poisons. Methyl or wood alcohol is a deadly poison whether drunk or inhaled in any considerable amount.

Ethyl alcohol is generally classed among the narcotic agents. It has, however, a primary stimulating effect upon the organs of the circulation, respiration and digestion, and also upon the brain. Its effects vary enormously with the dose and with the constitution of the patient. It is a food in the sense that about 90 per cent. is used up in the body and transformed into heat and energy, like sugar. It is not, however, a very good food under ordinary conditions, but it can be used as such, in daily amounts not over ℥ii by many people, after adolescence.

Alcohol is also, correctly, said to be a poison, though this may be said of almost any substance that is taken into the system, if used to excess. Alcohol, however, is a dangerous poison to a minority of people, either because they cannot use it temperately, a small dose leading them at once to excesses, or because it excites and disagrees with the functional activities of the system. Alcohol is always a poison when taken in excess and in some people a small amount may be an excess. The degenerate types of nervous systems are very sensitive to alcohol, and usually made worse by it, so that the use of it by degenerates tends to make them worse and extinguish this group of the race.

On the other hand, alcohol by intemperate use, can lead to an acquired degeneracy in healthy constitutions. History shows that both those races which indulge excessively in alcohol and those which do not use it at all, either degenerate or do not progress. Alcohol causes a small percentage of epilepsy through inheritance, and a still smaller percentage of epilepsy among its users. It is a large factor in the production of acute mental disturbances, such as delirium tremens, but not a large factor in causing degenerative insanities or imbecility or idiocy. In my own experience, the percentage of alcoholism in the ascendants of the insane, imbecile and idiotic, is about 5. The ratio

of insanity to the population is about the same in prohibition as in other states in this country. In persons who inherit degenerate constitutions, that is to say, in persons with a weak and unstable nervous system, alcoholism is often only an expression of this instability, not a cause of it. For it is a sign of degeneracy to drink alcohol to excess or to have a morbid susceptibility to its effects.

Alcoholism and habits of alcoholic excess in this country almost always develop before the age of thirty, and if drinking were prohibited to those below that age, there would be but little trouble. Wine drinking, if we except champagne drinking, is almost never a cause of alcoholism in this country, and is a much less important factor than beer. This is largely, no doubt, due to the fact that wine is relatively little drunk in America. Beer and ale, if used in excess, are most injurious forms of alcoholic beverage, except to out-door workers, because they lead to digestive and metabolic disturbances. It would be wise to forbid the use of alcohol to those under thirty, except in the form of light wines or beer, and those only to workmen and people who live out of doors. Even after this age it should only be given to the certified immunes, that is, to those able to drink moderately.

It follows that, theoretically, at least, the sale of alcoholic liquors should be controlled by the Boards of Health.

It would not be wise to abolish the use of alcohol altogether from civilization until we can be sure it has no good function, or until we can be sure that its abolition would not be followed by the use of more injurious substitutes. It is impossible to deny that civilization has satisfactorily advanced, despite alcohol, and has shown its finest types among those nations who have used it most. Owing to the fact that sanitation and civilization have developed a rather more delicate type of nervous organization, and to the fact that sanitary care has kept more of the unstable and weakly alive, we cannot use alcohol as freely as formerly. Therefore, alcohol has got to be used more and more carefully, and under closer supervision and perhaps finally will have to be abolished absolutely from general use.

Arterial sclerosis is a condition brought about by many of the factors that have been here enumerated as causing nervous disease. Its special and direct importance will be shown in the special chapters.

Reflex Causes.—Among other causes are local disease of viscera, such as renal, uterine, and ovarian diseases, dyspeptic and liver disorders, visual and auditory troubles. Reflex irritations are distinctively exciting causes, but with few exceptions they cannot cause a nervous disease unless there is a predisposition to it. They may, however, cause many distressing nervous symptoms, such as pain, spasm, and even convulsion.

CHAPTER III

GENERAL PATHOLOGY

The following is a list of the forms of disease which affect the nervous system:

1. Malformations; incomplete development, or agenesis; defective development, or dysgenesis.

2. Hyperæmia, anæmia, hemorrhage, œdema, and arterial and venous diseases.

3. Degeneration and atrophy, softening, sclerosis, classed as regressive processes.

4. Inflammations.

5. Tuberculosis and syphilis.

6. Tumors and parasites; 4, 5, and 6 being classed as progressive processes.

7. Nutritive and functional disorders, including disorders associated with metabolic and glandular defect, such as acromegaly and exophthalmic goiter.

The pathology of most of the above types of morbid processes will be given elsewhere, and does not call for discussion here.

Degeneration and Sclerosis.—By degeneration is meant in pathology a gradual death of the nerve-cells and fibres, or, in other words, of the parenchyma of the organ. The cells swell up, become granular and fatty, and then either break up and become absorbed or enter into a condition of a dead coagulum (coagulation necrosis). Degenerations may be acute or chronic, primary or secondary.

Acute neural degeneration causes a condition known as *softening* or *necrosis*. It is due to cutting off of vascular supply, direct injury, and to necrotic and inflammatory poisons attacking the neurons. Acute degeneration may be followed by a reparative process, which is called a reparative or reactive inflammation, and which ends, perhaps, in producing a cicatrix or sclerosis.

Chronic degeneration is a slow neuronic death and is accompanied and followed by a proliferative process which results in the production of connective tissue and sclerosis or gliosis. Degeneration is by some writers classed as a degenerative or *parenchymatous inflammation*.

Sclerosis is a process of connective-tissue proliferation, as a result of which the normal or injured parenchyma is supplanted by neuroglia

and fibrous tissue. The word sclerosis is usually employed in describing degenerative diseases, though it indicates the result rather than the primary nature of the process. In the nervous system there is often an increase or proliferation of neuroglia tissue in the processes of degeneration. Exactly how large a factor this is cannot yet be said. In one form of sclerosis—multiple sclerosis—the process of neuroglia proliferation seems to be the primary one, nerve-cell destruction following. Hence this type of sclerosis is classed with the proliferative inflammatory processes.

Degenerations are caused by certain poisons, such as arsenic, phosphorous, lead and the poisons of infectious disease. Degenerations also result from obliterating arteritis, such as occurs in old age or from humoral poisons. Degenerations sometimes are due apparently to an inherent defect in the cell nutrition—a premature death of it; and to causes yet unknown. The question as to whether certain scleroses are forms of productive inflammation or of chronic degeneration is one that has been much debated in the past. It is quite certain now that most of the so-called chronic inflammations of the nervous centres are really degenerative processes, and that the primary trouble is in the parenchyma, and not in the connective tissue.

Gliosis.—When the pathological process is the result of a proliferation of neuroglia, not of connective tissue, it is called gliosis.

Nutritive and Functional Disorders.—Under this head are included defects due to heredity, to disorders of the blood and blood-glands, to defects in metabolism, to poisons, extrinsic and autochthonous, and to local diseases.

CHAPTER IV

GENERAL SYMPTOMS

When the nervous system is disordered it produces various symptoms, which are classified and receive names according to the parts affected and the kind of change present. The general name given to any kind of morbid nervous state is *neurosis* and the general name for any morbid mental state is *psychosis*. When the neurosis affects the motor sphere, whether in the brain or cord or nerves, it is a motor neurosis; when the sensory parts are disordered we have a sensory neurosis. In the same way we have trophic, thermic, vasomotor and secretory neuroses.

The symptoms of nervous disease can be to a large extent divided in accordance with the kind of disturbances present. Now a function can be disordered in three ways. It may be exaggerated, lessened, to the point perhaps of entire loss of function, or it may be perverted. In order to indicate this, certain Greek prefixes are used. They are "hyper," which means excess; "hypo," meaning diminution; "a" or "an," indicating entire loss; and "para," meaning perverted. Thus we have, for example, hyperæsthesia, or excessive sensibility; anæsthesia, or loss of sensibility; and paræsthesia, which means perverted sensibility.

Finally, nervous symptoms are often spoken of as objective or subjective. The former are those symptoms which can be seen or directly noted by the physician without depending on the patient's statements. The subjective symptoms are those which are felt by the patient, but give no outward sign. Thus headache is a subjective symptom, paralysis is an objective one.

So far we have been grouping together only like kinds of symptoms; but it happens that one nervous *disease* may have quite different kinds, some being motor, some trophic or sensory. Thus nervous diseases practically are to a considerable extent classified not functionally but anatomically; and we have spinal cord and brain diseases, gastric and sexual neuroses, and so on.

Nervous *symptoms*, however, are always grouped together in accordance with the physiological function disturbed. So that we have the following tabulation (see also Fig. 17):

1. Mental and cerebral.
2. Motor and reflex.

3. Sensory (general, and special sense neuroses).
4. Trophic.
5. Vasomotor (angioneuroses).
6. Secretory (secretory neuroses).

Combinations of these groups of symptoms may affect various organs. They are called mixed neuroses. Combinations of mental and nervous symptoms form psychoneuroses.

The particular symptoms which nervous diseases cause will be described and recorded under the several heads given above.

1. **The mental symptoms** include all those found in insanity, idiocy, and imbecility, and will not be given in detail here. The common symptoms met with by the neurologist are mental irritability, depression,

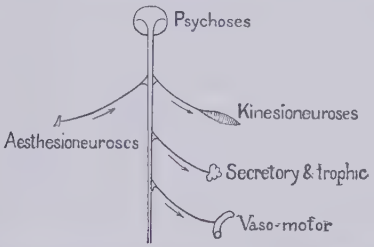


FIG. 17.—Diagram illustrating the principle of the classification of nervous symptoms, aside from special sense neuroses.

emotional excitement, morbid fears, volitional weakness and lack of self-control, persistent or fixed ideas, weakness of memory and of power of concentration, and a tendency to hypnotic and somnambulistic states.

Certain symptoms due to disturbance of brain function are called *cerebral* or *cerebellar*. They include such conditions as vertigo, disorders of equilibrium, stereognosis, aphasia, cerebellar ataxia, and many symptoms connected with the special senses, which will be described in the special chapters later.

2. **Motor Symptoms.**—The symptoms of disordered motility are as follows:

(*A*) *Symptoms of exaggerated or perverted motility.*

Tremor: (*a*) Fibrillary and wave-like movements of muscles (myokymia).

(*b*) Tremor proper.

Rhythmical spasm, athetosis.

Convulsion.

Myoclonia, a general name for muscular twitchings and including the choreic and tic spasms.

Hypertonia and contracture.

Forced and associated movements.
Ataxia and asynergy.
Exaggerated reflex conditions.

(*B*) *Symptoms of lessened motility.*
Paralysis and paresis, myasthenia, muscular atrophy.
Loss of reflexes, superficial and deep.
Hypotonia or lessened muscular tonus.

The particular characteristics of these different symptoms will be best shown in the description of the special diseases, but a brief account will be given here.

Tremor is the result of a disorder in the tonic innervation of muscles, and the cerebello-mid-brain mechanisms are important factors in many types. Muscles are kept normally in a state of slight tension with rhythmical impulses passing down at the rate of about twelve per second. When the rhythm and force of these normal impulses are interfered with we have tremor. The simplest form of tremor is one in which the normal rhythmic impulses have an apparently exaggerated force. This causes a *fine tremor* of eight to twelve vibrations per second (Fig. 19). When there is an interruption to some of the impulses we have a *coarse tremor*. Here the vibrations are four to eight per second caused by a partial or complete dropping out of the alternate impulse. Various technical names are used in describing the tremors. We have the fine and coarse, as described *Intention tremor* is one that occurs on voluntary movement, and is opposite in kind to the *passive tremor* or tremor of rest, which does not increase on voluntary effort. Tremor is sometimes of a coarse, jerky, and inco-ordinate character, and these words are then used to indicate it.

Fibrillary tremor or myokymia is a fine twitching of the individual strands or parts of muscles, and occurs usually when they are wasting from toxic or neurotrophic influence. Closely related to it are the wave-like contractions of the muscle-body without motor effect.

Convulsions consist of abnormal and exaggerated muscular contractions occurring in rapid succession. Convulsions may be *clonic*, *i.e.*, the muscles rapidly and alternately contract and relax in an exaggerated and irregular way; or they may be *tonic*, *i.e.*, contracted suddenly and steadily for several seconds or even minutes. When a tonic muscular contraction is painful it is called *cramp*. Convulsions may be co-ordinate. In this case the patient moves the limbs and body in a more or less purposeful way. He throws himself about the bed, jumps, kicks, strikes, tears the clothes, etc. Convulsions are usually accompanied with loss of consciousness.

Myoclonia is a term used to indicate muscular twitchings of various types, such as those of chorea and the "tics."

Choreic movements are sudden jerking, twitching movements of different groups of muscles. The movements are purposeless and are not under control of the will. *Convulsive tic* is a form of spasmodic movement confined to certain groups of muscles which work together for a common purpose, like those of the face, or eyes, or larynx. The movements in the "tics" are definite in character and are limited to muscles physiologically grouped for a special function. Thus we have tics of the muscles of expression, or of respiration, or speech, or locomotion.

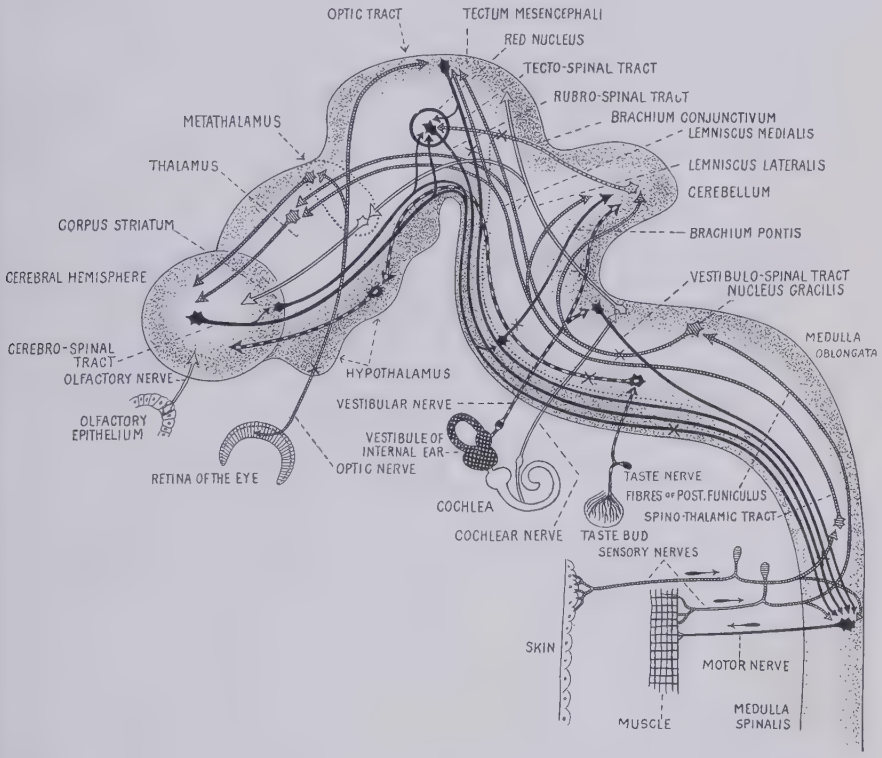


FIG. 18.—Showing in detail the neuronic architecture of the nervous system in the human embryo. (*Cunningham.*)

Athetosis is a name given by Hammond to a peculiar form of movement characterized by slow, successive flexion, extension, pronation, and supination of the fingers and hand and arm, or of analogous movement of the toes and feet. The motion rarely ceases in waking hours except for a short time. The contractions are forcible, steady, and even, and sometimes painful. The hand assumes characteristic positions.

A *contracture* is a tonic muscular contraction of long duration, *i.e.*,

days or months. A contracture may be functional or organic; and in order to test this, one must find whether it ceases during sleep or under an anæsthetic; if so, it is functional (see Hysteria). It may be paralytic; *i.e.*, due to paralysis of opposing muscles, or it may be due to a continuous spasm.

In forced movements the patient suddenly and involuntarily is thrown forward, sideways or whirled about in various ways.

Associated movements are those which occur involuntarily in a limb or muscle at rest when the corresponding limb or muscle is moved on the opposite side. Thus in hemiplegia the movement of the normal arm may excite a movement in the one paralyzed. The patient is given a piece of chalk in each hand, and each hand is placed upon a blackboard lying on the table; attempts at drawing lines with the sound arm cause movements of a similar kind, but less perfect, on the paralyzed side.

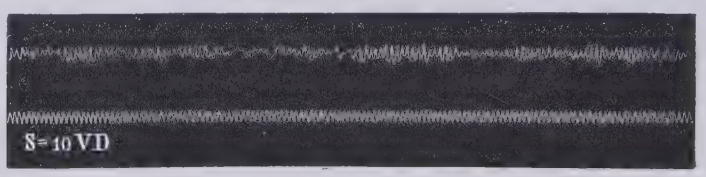


FIG. 19.—Diagram of a fine tremor. Ten of the divisions on the lower line equal a second.

Myoidema is a tonic spasm of a part of a muscle. It is produced by a sharp blow upon the muscle. This causes the muscular fibers to bunch up into a small tumor for several seconds. Its presence may indicate rapid muscular wasting from exhausting disease, or a toxic state.

Idiopathic muscular spasm is a phenomenon of a similar nature. When the belly of a muscle is struck with a dull instrument, a welt of contracted muscle appears and lasts several seconds. It indictes an exaggerated muscular irritability.

B. Symptoms of Lessened Motility

Paralysis or akinesis is a loss of motor power. Monoplegia is a condition in which one limb is paralyzed; hemiplegia one in which one-half the body is paralyzed; and paraplegia one in which the two lower limbs are affected. Sometimes a double hemiplegia or diplegia occurs. The term paralysis is sometimes used to indicate loss of any kind of function, as paralysis of sensation or secretion.

Paresis is a term used to indicate a partial paralysis. It is not

to be confounded with the term general paresis, which is a form of insanity.

The Reflexes.—When an impulse, started in an afferent nerve, reaches the spinal cord or medulla and is thence reflected upon an efferent nerve, the result is called a simple *reflex action.* The process is an involuntary one. It ordinarily occupies one-tenth to one-twelfth of a second. The afferent nerve may be an ordinary cutaneous sensory nerve, or it may be a special nerve whose function is to excite reflex action. These latter nerves are called excito-reflex. This kind is principally supplied to the viscera.

In neurology we have to do with three kinds of simple reflexes:

The skin or superficial reflexes.

The deep reflexes.

FIG. 20.—Babinski reflex.

The visceral reflexes.

The idiopathic or direct muscle response is not, strictly speaking, a reflex.

All these may be exaggerated, modified, lessened, or absent. Further description of the reflexes will be given under the head of diagnosis.

The simple reflexes are combined to form compound or higher and more complex reflexes, which underlie *automatic actions.*

3. **Sensory Symptoms.**—The sensory functions include all those belonging to the nerves of general and special sensation. Sensory nerves have a part in reflex action and in the inhibition of motor and other functions. The nerves of special sense when deranged show various phenomena, which will be described in more detail later. In general there may be depression or loss, increase or perversion of their function. In accordance with this we have:

Anæsthesia, which is a loss of tactile sensibility.

Analgesia, a loss of sensibility to pain.

Thermo-anæsthesia, a loss of sensibility to temperature. There may be loss of cold sense, of heat sense, or, as is usually the case, of both senses.

The term anæsthesia is often used with a general meaning to indicate loss of all forms of sensibility. Anæsthesia in this sense is a symptom referred to the skin, bones, mucous membranes, special senses or viscera. The muscles have two kinds of sensibility: a sensibility to pain and pressure and a special muscle sense. Anæsthesia of the pain-pressure sense of muscle is called loss of muscular sensibility or muscular analgesia. Anæsthesia of the special muscle sense is one of the factors in causing a symptom known as ataxia.

Ataxia is a symptom due to loss of the special sensibility of the muscle, articular surfaces and tendons, causing irregular and inco-ordinated movements. This special sense, sometimes called deep sensibility, informs the individual of the degree and strength of muscular movements, and by it definite and co-ordinated movements are made possible. The weight of objects and position of the limbs are also determined by it. In *static ataxia* there is loss of the power to preserve perfectly the equilibrium when standing. In *locomotor* or *motor ataxia* there is loss of power to co-ordinate the limbs properly in motion. In both these conditions there is also usually a loss of power to appreciate weights or the position of the limbs.

Cerebellar ataxia is a form of inco-ordination and disturbed equilibrium due to disease of the central organ of equilibration, viz., the cerebellum. Cerebellar ataxia is due to disturance of (*a*) the equilibrium mechanism, (*b*) and of certain co-ordinating and tonic mechanisms which enable one to move the segments of the body in proper degree and rhythm. Their disturbance causes symptoms called *asynergy*, *adiokokinesis*, and *hypermetria*. These will be described later in connection with the diseases of the cerebellum.

Asterecgnosis.—The ability to recognize the form of objects held in the hand or on other parts of the body is called *stereognosis*, and its loss *astereognosis*. It is a perceptive process, due to the fusing and elaboration of cutaneous and deep sensations, especially the latter. Astereognosis is present in peripheral or spinal lesions where there is complete tactile anæsthesia, and where, even with good tactile sense, there is loss of deep sensibility. As complete tactile anæsthesia is rare in cerebral lesions, the cause of astereognosis here may be due to injury of the center for deep sensibility or of perception of form. The lesion in such cases is localized in the parietal lobe.

The ability to recognize not only the form but the nature of an

object is called *symbolia*, and its loss *asymbolia*. The recognition of the nature of an object is only a higher elaboration of the preceptive work than that in which simply form is recognized. But the nature and location of the process are the same, practically, as for stereognosis.[1]

Hyperæsthesia is an excessive sensibility to touch, contact, and other sensory stimuli.

Hyperalgesia is excessive sensibility to pain, and is nearly identical with tenderness.

Dysæsthesia is simply disagreeable paræsthesia.

Paræsthesia is a term applied to all the morbid general sensations except pain. The paræsthesias include such feelings as numbness, prickling, formication, flushing, burning, itching, coldness, tickling, various peculiar visceral sensations. Ordinarily in speaking of paræsthesiæ, however, we refer to such feelings as numbness, prickling and creeping.

Delayed sensation is a symptom in which an appreciable time exists, usually one or more seconds, between the time of applying a stimulus and its appreciation in consciousness. Normally a tactile sensation can be felt and responded to in less than one-tenth of a second.

Transferred or *referred* or reflex sensations are those in which the irritation is made at one point and felt at another. Thus an irritation in the stomach causes a pain felt in the forehead. The whole class of so-called reflex pains are really transferred sensations, since in reality there is no reflex action in the process, as will be seen later. *Allochiria* is a peculiar form of transferred sensation, in which an irritation applied on one side of the body is referred to a corresponding point on the opposite side.

4. **Trophic Disorders.**—These consist, so far as relates to neurology, chiefly of hypertrophy and atrophy of nerves, muscle, cutaneous and mucous tissues, joint degenerations and various skin eruptions. The trophoneuroses, if they affect joints, are called arthropathies; if muscles, atrophies, hypertrophies, and dystrophies; or if with atrophy there is a great substitution of fat, the condition is known as lipomatosis. When nerves are affected there results degeneration. Trophoneuroses of the skin produce various symptoms, such as herpes, pemphigus, pigmentation, leucoderma, alopecia and bed-sores. Trophoneuroses so called are not due directly to lesions of trophic nerves, but indirectly to disturbance of vascular, secretory, sensory and motor nerves.

5. **Vasomotor and Secretory Symptoms.**—The nerves supplying the blood-vessels and secreting glands work together and are usually disordered together. Separate disturbances of the vessels and glands,

[1] The terms *tactile amnesia* (Burr) and *gnosia* and *agnosia* are used for these or similar perceptive processes.

however, occur. *Angioneurosis* is the term given to disorders of the vasomotor centre and nerves. *Angiospasm* is a condition in which there is increase of vasomotor tone and spasmodic contraction of the muscular coats of the arteries. *Angioparalysis* represents the opposite condition. Such disorders affecting the skin are shown by pallor and coolness or by flushing and heat. *Angioataxia* is a condition of variability and irregularity in the tonus of the blood-vessels.

The *secretory neuroses* affect the functions of the skin, mucous membranes and special glands. *Hyperidrosis* is an excessive sweating. *Anidrosis* is excessive dryness. *Paridrosis* is a perversion of secretion in which peculiar odors or colors are noted. *Hæmidrosis* is the term applied to bloody sweating.

The secretions of the internal organs are controlled by nervous influences, and their special disturbances often form part of the symptoms of nervous diseases. Thus we have watery diarrhœa in Basedow's disease, and a peculiar membranous discharge from the bowel in asthenic states.

The glands of internal secretion and particularly the thyroid and pituitary glands, have perversions of function which apparently lead to serious nervous symptoms, which will be described under the head of exophthalmic goitre, acromegaly, and dyspituitarism.

CHAPTER V

DIAGNOSIS AND METHODS OF EXAMINATION

The diagnosis of a nervous disease may be simply a clinical one; that is to say, one may recognize it as belonging to a certain known and definite group of symptoms. Thus, in recognizing the phenomena of epilepsy, one makes a clinical diagnosis. In other cases, and especially in all organic nervous diseases, the physician must make in addition a local and then a pathological diagnosis. That is, we must determine the seat and nature of the disease.

A diagnosis is made by first getting all the obtainable facts in the patient's past history, then by learning from him all his subjective symptoms, and finally by making an examination according to the technical methods to be here described. In examining a patient, it is imperative that a careful search for diseases outside the nervous system first be undertaken. Then the morbid nervous phenomena should be investigated. The physician should make it an invariable rule to make this examination in a certain fixed and systematic manner. The best method is first to get the family and personal history, and then to go over the mental, cerebral and special nervous functions serially in the way indicated under the description of general symptoms.

In addition to the routine methods we have often to make special tests to determine the existence of foreign bodies, fractures, tumors and disturbances of the blood, urine and cerebrospinal fluids.

In investigating the family history, it is often necessary to make very direct and probing inquiries, for patients are, as a rule, inclined to forget or ignore the existence of nervous and mental disease among relatives. The existence of consumption and inebriety, epilepsy and syphilis in the direct line are very important facts; so also are those concerning birth. The patient should be questioned closely as to his previous diseases, especially syphilis; also as to his habits in relation to sexual indulgence, indulgence in alcohol, and smoking. In women, the tea habit should be inquired into. The patient may be allowed to tell his own story first. Proper queries should be put to supplement this, and finally the patient should be asked to state those symptoms which to his mind are main and dominant.

We will now go over the above points in detail.

1. **The Physiognomy,** complexion, and general nutrition are first noted. Many nervous disorders are compatible with a very healthy

appearance, and patients often make the introductory apology, "I don't look like a sick person." An anxious look, restless manner, and excited or diffident speech, however, often show something wrong. The nervous trouble is usually serious in reverse proportion to the voluble anxiety of the patient to make his condition exactly understood. The character of the gait may reveal at once the nature of the malady. The dropped foot and flaccid swing of the leg in poliomyelitis and neuritis, the stiff shuffling march of paraplegia from myelitis, the waddling move-

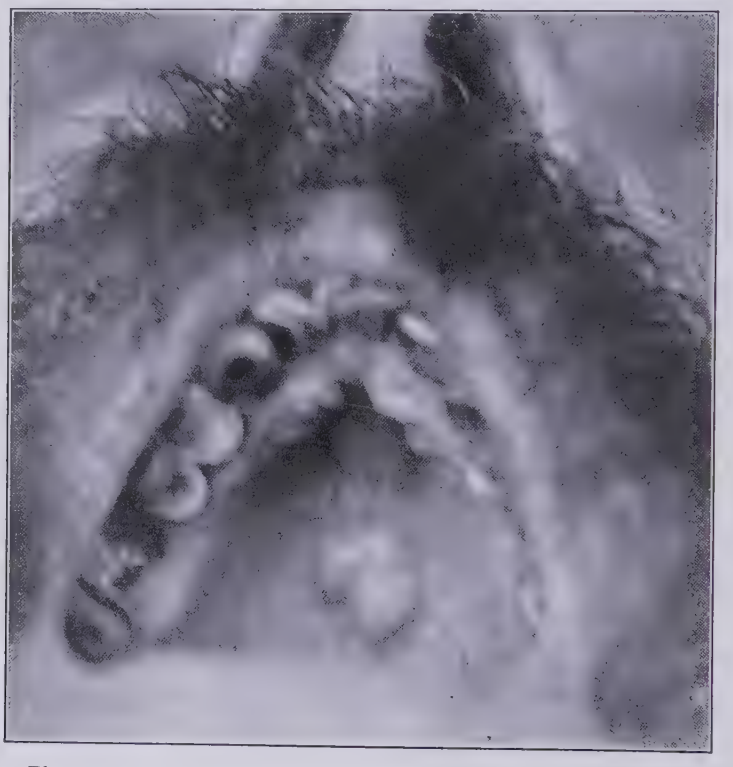


Fig. 21.—Photograph of roof of the mouth of a drunkard showing torsus palatinus.

ments of juvenile muscular dystrophy, and the bent head and careful stamp of locomotor ataxia are almost of themselves diagnostic:

Et verus incessu patuit morbus.

The speech also often betrays the malady. The physician soon gets to recognize not only the striking symptoms of aphasia, but also the weak piping of paralysis agitans, the stumbling enunciation of paresis, and the peculiar dysarthrias of multiple sclerosis and bulbar palsy. As a rule, the occurrence of speech difficulties in adults is significant of organic and often serious disease.

In the chronic and constitutional nervous maladies note should be made of the marks of degeneration or the *neuropathic constituent.* The nature of this condition has already been described under the head of hereditary causes of nervous disease. The existence of degeneration implies an imperfect, unusual, or unbalanced development. The condition is usually shown in some nervous or mental peculiarity or defect in the individual, and degeneracy, as ordinarily understood, implies a neuropathic or psychopathic state. But degeneracy may also mean only a lessened vital resistance to certain forms of infection or injury, as, for example, in persons of a tuberculous tendency, who often have marks of degeneracy. These marks are called the stigmata of degeneration. They are of three kinds: anatomical, physiological and mental.

Anatomical stigmata:

Cranial anomalies, *e.g.*: Asymmetry of cranium. Microcephalus. Peculiar shape of skull, trigonal, scaphocephalic, plagiocephalic. Facial asymmetry and excessive prognathism. Large jaws.

Deformities of the thorax, such as the pigeon-chest, the funnel-chest, the narrow barrel-shaped chest with rib set too obliquely on the spine.

Deformities of the palate and uvula, including high narrow arch and the torus palatinus.

Anomalies of the teeth, tongue and lips.

Anomalies of the eyes: narrow palpebral fissure, muscular insufficiency, excessive astigmatism, nystagmus.

Anomalies of the ears: badly placed, ugly shapes, asymmetry, adherent or lobeless ears, markedly conchoidal ears.

Anomalies of the limbs, genital organs and body generally.

Anomalies of the skin, excessive hairiness or absence of hair.

The most imporant of the anatomical stigmata are deviations in the symmetry and shape of the skull, defects in the palate and underjaws, badly shaped ears, badly set teeth, badly set ribs and a generally weak and badly developed body. Stress is laid upon the skull because its development corresponds with that of the brain. The palatal stigmata are in general those which make the cavity of the mouth smaller, it being the fact that the mouth cavity increases in size as we ascend the vertebrate series. Abnormal palates are found in about 10 per cent. of normal people (Charon) and in from 46 to 80 per cent. of abnormal. The high, narrow palate is one oftenest seen by myself. The torus palatinus or longitudinal ridge on the hard palate is significant if it is well marked (Fig. 21). The importance of defective ears is based upon comparative observations. They are found in from 20 to 64 per cent. of more or less abnormal persons.

While many of the stigmata have no significance in themselves, yet the presence of a number of them is of great importance, for neuroses or psychoses developed among this class have a much more unfavorable

prognosis. It is especially among neurasthenics, epileptics, severe forms of hysteria and in the insanities that these signs are to be looked for and studied. Among normal men about two or three anatomical stigmata are often found; among lunatics, criminals, abortive types of paranoia and primary forms of neurasthenia the number is much greater.

The physiological stigmata include tremor, tics, nystagmus and hereditary defects in the muscular system leading to atrophies. Excessive or defective sensibility of the cutaneous and special senses, defects in speech, perversions of the sexual and other instincts are to be classed here. A diminished resistance to nervous and emotional strain is a most frequent physiological mark of neuropathy. Allied to this is the excessive sensibility to or craving for the action of tea, coffee, tobacco and alcohol.

The mental stigmata include all those factors that make up the erratic, unbalanced and morbidly emotional individual. The specially morbid note in these persons is an excessive egotism, an intense self-consciousness, often with peculiar disturbances of the sense of personality.

Mental retardation is often an important factor in the examination of a nervous patient. This is especially true in epilepsy and in the psychoneuroses, and in the neuroses of childhood. The Binet-Simon and certain adult tests for intelligence are to be employed here.

Physical defects due to disturbance of the functions of the *glands of internal* secretion may need investigation, and this would come in a measure under the head of physiognomy, and nutrition.

2. **Investigation of Symptoms of Disordered Motility.**—In studying the attitude, expression, gait and speech, some notion of the condition of the motor functions has been obtained. Special disturbances of the various parts must then be investigated.

Paralysis.—The patient is made to move the arms, legs, trunk, head, facial muscles, eyes and tongue, etc., in all possible ways. If paralysis is found the degree of it in some groups of muscles can be measured by dynamometers. The ordinary hand dynamometer measures the degree of paralysis in the flexors. The average power of pressure on the dynamometer is, for an adult, 40 to 50 kilograms for the right hand, and 3 to 5 kilograms less for the left. A woman has about two-thirds of the power of a man. A good idea of the degree of paralysis can be got by making the patient take the physician's two hands with his own and squeeze each at the same time. A malingerer or hysteric will often in this way unconsciously press much harder than he is aware. The physician's own ingenuity will suggest various ways of testing the strength of the leg and thigh muscles, such as making the patient rise on one toe, climb upon a chair, push against an object with his foot, etc.

TABLE OF CRANIAL MEASUREMENTS

	Adults		Physiological Variation	New-born		End of 1st yr.		1st to 7th yr.		10th year		7th to 18th years	18th to 24th years.	End of 12th yr.	
	M.	F.		M.	F.	M.	F.	M.	F.	M.	F.				
1. Greatest circumference	52.0	50.0	48.5 to 57.4	34.0	34.0	42.0	42.0	34 to 46	.....	49	47	46.0 to 49.5	49.5 to 52.25	.....	Taken around glabella and maximal occipital point.
2. Binauricular arc	32.0	31.0	28.4 to 35.0	20.0	20.0	25.5	25.0	.....	27	.....	.....	.....	.....	30	Measured from *B* over through bregma to *B*, or opposite ext. aud. meatus.

These figures are too low for children of the educated classes in this country by at least 1 cm. The microcephalic skull has a capacity below 1350 c.c.; the mesocephalic, a capacity between 1350 and 1450 c.c.; and the megacephalic, a capacity over 1450 c.c.

Some paralysis being discovered, we try to determine its degree and type. Paralyses are of four types:

1. An upper neuron type, in which the lesion involves the cerebrospinal motor neurons. The paralysis is then as a rule hemiplegic, and accompanied with spasticity and exaggerated deep and lessened skin reflexes; it is not accompanied with atrophy of the muscles, involved or with degenerative electrical changes.

2. A lower neuron type, in which the lesion involves the anterior cornual cells or anterior roots of the spinal cord, or the corresponding parts of the brain stem (pons-medulla). The paralysis is localized in the muscles supplied by the injured neurons; it is flaccid and associated with atrophy and the electrical reactions of degeneration.

3. A neural, or mixed nerve type of paralysis, in which the symptoms are like those of a lower neuron type plus sensory symptoms, such as pain and anæsthesia.

4. A psychic type of paralysis, in which the paralysis depends upon a mental state. Here the paralysis may be hemiplegic, paraplegic or monoplegic but it is not accompanied with spasticity, or atrophy, and it is generally accompanied with anæsthesia.

Tremor is usually most noticeable in the hands and is tested by making the patient hold out the hands and arms at full length, spreading out the fingers at the same time. To determine whether the tremor increases on volitional movement, give the patient a full glass of water, let him hold it out for a moment, then bring it to his mouth slowly. If the tremor increases with this movement it is called "intentional." As a general rule, the tremor of organic disease is coarse in type and increased by volitional movement, and ceases during rest of the extremity. Functional tremors are usually fine in type and continuous, except on complete rest and relaxation. In most forms of tremor the hand and arm shake as a whole. In other forms the tremor involves only the fingers or hand or forearm and hand. Such tremor is called segmental. It is especially seen in paralysis agitans. As I have already said, tremor may be fine or coarse, *i.e.*, four to six or eight to twelve per second. To determine this accurately a special apparatus is needed; but one can with a little experience determine this fairly well by observation alone.

While coarse tremor as stated is usually a sign of organic disease or of paralysis agitans, it occurs also in hysteria and grave conditions of alcoholism. Tremor that is hardly observable by the eye can be felt by placing one's hand against the extended fingers of the patient.

Tremor of the tongue and lips and facial muscles must be carefully looked for. It is tested by making the patient close the eyes tightly and show the teeth or protrude the tongue. Facial tremor if very marked usually indicates a serious condition of nervous exhaustion, alcoholic

poison, or some other toxæmia or paresis. Tremor of the whole head due to the neck muscles must be distinguished from secondary shaking of the head due to a tremor of the trunk.

Finally, it is to be remembered that tremor is a rhythmical disturbance and it should be distinguished from the jerky irregular ataxic movements of cerebellar-mid-brain disease. These always cease entirely when the patient is lying on his back and making no movements.

Nystagmus is a form of tremor indicating cerebellar or mid-brain disturbance.

Choreic movements, tics, associated and forced movements, and convulsions are the expression of certain diseases and are described elsewhere.

The Examination of the Reflexes.—These are of three kinds: (1) the superficial or skin, (2) the deep, (3) the visceral.

1. A skin or superficial reflex is produced by scratching, pinching or irritating the skin. The result is a contraction of the muscles supplying the parts near or under the irritation. The skin reflexes which can be ordinarily brought out are the anal, bulbo-cavernous, plantar, cremasteric, epigastric, abdominal, scapular, palmar, and certain cranial reflexes.

The anal reflex is brought out by scratching the perineum. It causes a contraction of the sphincter ani.

The bulbo-cavernous reflex is brought out by placing a finger on the urethra just back of the scrotum, and pinching or pricking the glans penis. This causes a contraction of the bulbo-cavernous muscle.

The plantar reflex is produced by tickling or scratching the soles of the feet. This causes usually, when carefully done, a slight flexion of the toes. In many cases there is, however, no response. In irritable persons and children there is a sudden dorsal flexion of the foot, and often a contraction of the inner hamstring muscles. In pathological conditions involving the pyramidal tracts of the cord and the motor centres and tracts in the brain there is a dorsal extension of the great toe, sometimes accompanied with a fan-like spreading out of the other toes. This is called the Babinski reflex (Fig. 20). A similar phenomenon is produced by firmly compressing the muscles of the calf (Gordon reflex), and by causing the patient to draw his leg up sharply flexing the thigh on the abdomen and the leg on the thigh (Strumpell reflex); or by pressing and drawing a blunt instrument or the finger sharply down along the inner side of the tibia (Oppenheim reflex) or by scratching the leg behind the external malleolus (Chaddock reflex).

The *cremasteric* reflex is brought out by scratching the inner side of the thigh or the skin over Scarpa's triangle. It causes a drawing up of the testicle, not of the scrotum alone, on the same side.

The *abdominal* reflex consists of a contraction of the abdominal recti

muscles, caused by irritating the skin over the outer border of the rectus.

The *epigastric* reflex consists of a contraction of the upper fibres of the rectus, caused by irritating the skin of the same region higher up.

The *scapular* reflex consists of a contraction of scapular muscles, caused by irritating the skin over them.

The *palmar* reflex is produced by irritating the palms of the hands. It is obtained only in infants and the reaction is flexion.

The cranial reflexes are the *corneal* and *conjunctival*, a reflex contraction of the lids caused by lightly touching the cornea or conjunctiva with a bit of cotton or a camel's-hair brush; the *pupillary-skin* reflex, which consists of a dilatation of the pupil caused by scratching the skin of the cheek and chin; the *supra-orbital reflex* caused by striking a slight blow over the supra-orbital foramen; the *naso-mental* reflex, in which a contraction of the levator mentis is caused by a tap on the side of the nose.

Skin or Superficial Reflexes

Superficial Reflexes	Method of Exciting	Effect	Localization
1. The Supra-orbital.	A tap over the supra-orbital foramen	Orbicularis contracts	V–VII
2. Conjunctival reflex.	Touching cornea, or conjunctiva	Orbicularis contracts	V–VII
3. Naso-mental....	Blow on side of nose	Levator menti contracts	V–VII
4. Palatal.....	Touching palate	Palate contracts	IX–X
5. Pharyngeal.....	Touching posterior wall of pharynx	Pharynx contracts	IX
6. Skin-pupillary..	Pinching cheek	Pupil dilates	V–cervical sympathetic
7. Scapular.....	Stimulation of the skin over the scapula	Movement of shoulder-blade	C5–D1
8. Palmar.....	Irritation of the palm	Flexion of fingers	C8–D1
9. Epigastric.....	Stroking from nipple downward	Drawing in of epigastrium	D7–D9
10. Abdominal.....	Stroking side of the abdomen	Drawing in of abdominal wall	D9–D12
11. Cremasteric....	Stroking the adductor region of the thigh	Elevation of testis	L1–L2
12. Plantar reflex...	Stroking the sole	Dorsal or plantar flexion of toes	S1–S2
13. Bulbo-cavernosus.	Pinching dorsum of glans penis	Bulbous urethra contracts	S3–S4
14. Anal reflex.....	Pricking the perineum	Contraction of sphincter ani externus	S5

There are also palatal and pharyngeal reflexes. The superficial reflexes depend upon the integrity of the reflex spinal arc, and to a less extent upon the degree of cerebral influence. When present, they show that the spinal cord at the level through which the impulses travel is healthy. When absent, they do not necessarily indicate much of anything, for they vary in amount in different persons and at different ages being always more active in the young. In cerebral hemiplegia during and after the acute attack the abdominal reflexes are absent on the affected side. This reflex is also often absent in multiple sclerosis. The supra-orbital reflex is often absent in profound hemiplegia with coma.

Fig. 22.—Getting the knee-jerk.

The *deep reflexes* are sometimes called *tendon reflexes*, though this is not a strictly correct name, since they can be called out by striking periosteum or muscle as well as tendons. The deep reflex in all these cases is not a true spinal reflex, but is due to the direct effect of the concussion or the sudden stretching upon the muscle itself which is in a condition of slight tonus. Those who accept this view speak of the deep reflex as indicating the myotatic irritability or muscular tonus. The deep reflex implies also the integrity of a reflex arc.

The important deep reflexes are the patella-tendon reflex or knee-jerk; the ankle reflex or ankle-jerk; the biceps, supinator, pronator and triceps reflexes; the scapulo-humeral reflex; the jaw reflex or chin-jerk; the light (or pupillary) and so-called accommodation (or ciliary) reflexes; the oculo-cardiac reflex. These are all present in health except the jaw-jerk.

The reflexes are of diagnostic importance, for they are absent in some diseases, exaggerated in others, and in certain conditions new reflexes appear. These abnormal reflexes are the Babinski reflex, the Hoffman reflex, ankle clonus and certain pupillary reflexes.

The patella reflex or knee-jerk consists of a sudden contraction of the quadriceps femoris, vastus internus, and subcrureus caused by striking the patella tendon when the leg hangs loosely at right angles with the thigh. This reflex may also often be produced by striking

the lower part of the muscle itself. The activity of this reflex is increased if, at the same time that the blow is struck, a voluntary contraction of some other muscles is made by the patient. Usually the patient is told to pull on his clasped fingers or tightly shut the hands. This process is called the *re-enforcement* of the knee-jerk (see Fig. 22). Such re-enforcement can be caused by irritating the skin and by various sensory or psychic stimuli. The nerve-roots involved are those, in man, of the second and third lumbar segments. The peripheral nerve is the anterior crural. The essential muscle is the vastus internus.

The biceps reflex, a supinator reflex, and a pronator reflex are said by Babinski to be always present in normal persons. The first two are obtained by striking the lower tendon of the partly flexed biceps and the lower third of the supinator longus; the pronator reflex is obtained by striking the lower head of the ulnar bone. The triceps reflex or elbow-jerk is brought out by striking the triceps tendon while the arm is supported and the forearm allowed to hang down loosely at right angles to the arm. These reflexes occur in normal individuals.

DEEP REFLEXES

Tendon, Bone or Deep Reflexes	Method of Exciting	Effect	Localization
1. Pupillary reflexes	Expose to light; and test accommodation	Pupil responds to to light, and accommodation	I–III ciliary ganglion Pons
2. Jaw reflex.....	Tapping lower jaw which is relaxed half open	Jaw closes	V
3. Biceps.....	Tapping biceps tendon	Biceps contracts	C5–C6
4. Supinator longus	Tapping styloid process of radius	Supinator longus contracts	C5–C6
5. Scapulo-humeral	Tapping vertebral border of scapula near base	Teres minor, infraspinatus, etc., contract	C5–C6
6. Pronator.....	Tapping head of ulnar	Pronation.....	C6–C8
7. Triceps.....	Tapping triceps tendon	Triceps contracts	C7–Th1
8. Carpo-metacarpal	Tapping wrist	Fingers flex	C8–Th1
9. Knee.....	Tapping patellar tendon	Vastus internus, etc., contract	L3–L4
10. Ankle.....	Tapping tendo-Achillis	Calf muscles contract	S1–S2

The jaw reflex or jaw-jerk is brought out by having the patient open the mouth and leave the jaw relaxed. A flat instrument like a paper-

cutter is then laid on the teeth of the lower jaw, and if this is struck smartly the elevators of the jaw contract. The light reflex is tested by throwing a bright light into the eye; and the ciliary or accommodation response by making the patient look at a distant and then at a near object. The pupil normally dilates in the former case and contracts in the latter. When the light reflex is lost while the accommodation action remains, the condition is called the *Argyll-Robertson pupil.*

The reflex path is probably through the optic nerve, the primary optic centers, third nerve, ciliary ganglion and ciliary nerves.

The *oculo-cardiac reflex* is produced by pressing firmly on the ball of one or both eyes. This causes a slowing of the heart beat. The reflex is usually absent in locomotor ataxia.

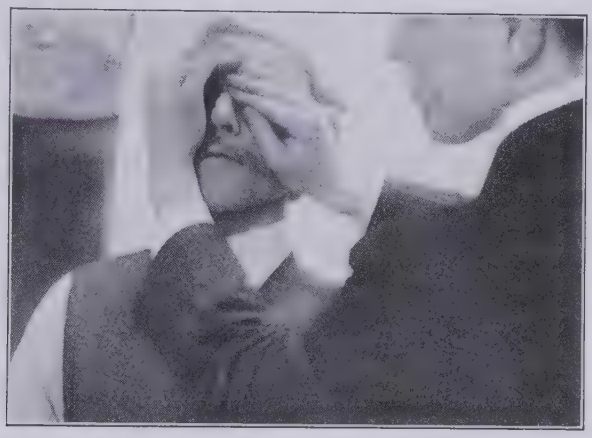


FIG. 23.—Testing pupillary reflexes, before an open window.

The Hoffman reflex.—A sharp snap of the last phalanx of the middle finger causes flexion of the index and thumb. Its presence generally indicates a pyramidal tract lesion.

Ankle clonus is caused by having the seated patient extend the limb and hold it rather firmly in a semiflexed condition. The physician takes the foot by the toe and heel and quickly flexes the foot on the leg. He thus suddenly stretches the calf muscles, and they undergo rhythmical contraction. This phenomenon does not occur in healthy people. It is found in involvement of the cortico-spinal tracts and it usually indicates organic disease of the cord. A pseudo-clonus sometimes occurs in which there are a few rhythmical contractions on sudden dorsal flexion of the foot, but the contractions soon subside. This is seen in exhaustion and toxic states and in hysteria.

The deep reflexes may be decreased, delayed, absent, or exaggerated. Their exaggeration is common and not of special clinical significance.

The absence of the knee-jerk or ankle-jerk is of great significance, indicating in persons who have no paralysis of the muscles, locomotor ataxia, neuritis or some toxæmia, such as follows diphtheria or exists in diabetes.

The Electrical Conditions in Disturbances of Motility.—These cannot be understood without some description of the methods of using electricity, and hence the technic of electrical examinations for purposes of diagnosis will be described under the head of treatment.

Examination of the Disorders of Sensation.—The object of examining the sensory functions is to see if they are exaggerated, perverted, delayed or lost, and to locate the extent of the disturbance. Patients differ greatly in their intelligence and power of description, so that great care must be taken in drawing conclusions as to sensory disturbances. In examining the skin and muscle-senses, the patient's eyes should be closed and he should be carefully told to answer promptly whenever he feels the stimulus. It is best to insist that he always reply in the same way, *e.g.*, using the word "now" the moment the sensation is felt. Many ingenious instruments have been devised, and I have described some of them, but for ordinary purposes a camel's-hair pencil or a bit of cotton wool and a pin answer very well.

Fig. 24.—Testing for ankle-jerk.

Loss of cutaneous and deep sensibility produces anæsthesias and these anæsthesias like paralyses have different characters in accordance with the series of neurons involved.

Anæsthesias, cutaneous and deep, may like paralyses be of different types in accordance with the part involved.

1. Upper neuron anæsthesias. If the upper sensory neurons of the cerebrum are affected the anæsthesia is never abslutely complete, and it is associated usually with a hemiplegia.

2. Middle neuron anæsthesias. If a lesion lies in sensory neurons of the cord or brain-stem the anæsthesia may be of a dissociated type; *i.e.*, there may be an anæsthesia to pain and temperature, but not to touch.

3. Peripheral neuron anæsthesias may be complete, or have various degrees of completeness, but they are not dissociated. Protopathic and

epicritic variations of cutaneous anæsthesia are all due to lower neuron and peripheral nerve involvement.

4. Psychic anæsthesias are rarely absolute. They involve the entire half of the body, or the entire body or one limb, and affect the special senses in a definite manner (see hysteria).

The *cutaneous sensations* are: (1) The tactile sense, which includes pressure and contact; (2) the temperature sense, which includes the heat sense and cold sense; (3) the pain sense. The first two are special senses, the last is a general sense.

To test the *tactile sense*, blindfold the patient and use the æsthesiometer. This is an instrument with two rather blunt points, which

FIG. 25.—Bringing out ankle clonus.

can be separated or approximated. A hairpin or two ordinary pins can be used in its stead. Its use depends upon the fact that the power to appreciate the contact of two points on the skin gradually approximated varies with the tactile sensibility of the patient. The tongue, finger tips and lips are the most sensitive points. The back, arms and thighs the least sensitive.

The following table shows the average distance at which two points are appreciated as such by an intelligent adult:

Tip of Tongue	1 mm. ($\frac{1}{25}$ in.).	Tip of toes, cheeks, eyelids	12 mm.
Tip of fingers	2 mm.	Temple	13 mm.
Lips	3 mm.	Back of hands	30 mm.
Dorsal surface of fingers	6 mm.	Neck	35 mm.
Tip of nose	8 mm.	Forearm, leg, back of foot	40 mm.
Forearm	9 mm.	Back	60–80 mm.
		Arm and thigh	80 mm.

The figures vary somewhat with the thickness or softness of the

skin and with the dullness or keenness of the nervous organization. If the distances are double those given above, it may be considered in most cases abnormal.

The sense of contact, which is a form of tactile sense, is tested by touching the skin very lightly with a hair or hair brush or bit of cotton. The sense of locality or power to localize a point on the skin that has been touched varies with the tactile sense and with the muscular sense. It is tested by placing the finger lightly on a given spot and telling the patient with closed eyes to place his finger on the part touched. He should come within 5 cm. In slight degrees of anæsthesia dependent upon disease of the sensori-motor areas of the cortex of the brain this is an important test. Further tests may be made by moving points along the skin and taking the patient to indicate the direction of the motion.

To test the pressure sense, one may use the baræsthesiometer, an instrument made with a spring scale measuring the amount of pressure made. A simpler way is to have the patient *rest the hands on a table* and then try and determine the weight of different objects. The lightest weight that can be appreciated on the hands or face is one of about 0.02 gram (gr. $\frac{1}{3}$). Differences of light weights of 1 and 5 grams and of 25 and 30 grams are about all that can be ordinarily appreciated by the skin. Much smaller differences, of 0.5 to 2 grams, can be detected if great care is used. Weighted rubber balls may be used in the foregoing test. I prefer to use differently weighted metal bodies held by a wire. Pressure sense is acute on the forearm and abdomen, where locality sense is feeble; also on the brow, temples and back of the hand. By the use of these tests held freely in the hand we test also the musculo-articular or deep sensibility.

The *temperature sense* is tested by test-tubes filled with hot and cold water, or by using hot and cold spoons, or roughly by breathing and then blowing on the part. A thermo-æsthesiometer may be used. This has a round, flat surface 1 cm. in diameter, and contains in its terminals thermometers by which the degree and differences in temperature may be noted. A small heated or chilled surface is appreciated much less easily than a large one.

The indifferent range where objects are felt to be neither warm nor cold is from 27° to 30°C. (80.6° to 86°F.). Fine differences (0.2° to 1.5°C.) are appreciated above the indifferent range. Lower down in the scale, differences from 1° to 1.3°C. (2° to 3°F.) are appreciable. It may be considered a morbid symptom if temperatures of 60° to 65°F. are not felt as cold, or temperatures of 86° to 95°F. are not felt as warm; also if between the ranges of 1°C. (32°F.) and 40°C. (104°F.) differences of 2°C. are not appreciated. A painful degree of sensitiveness to heat

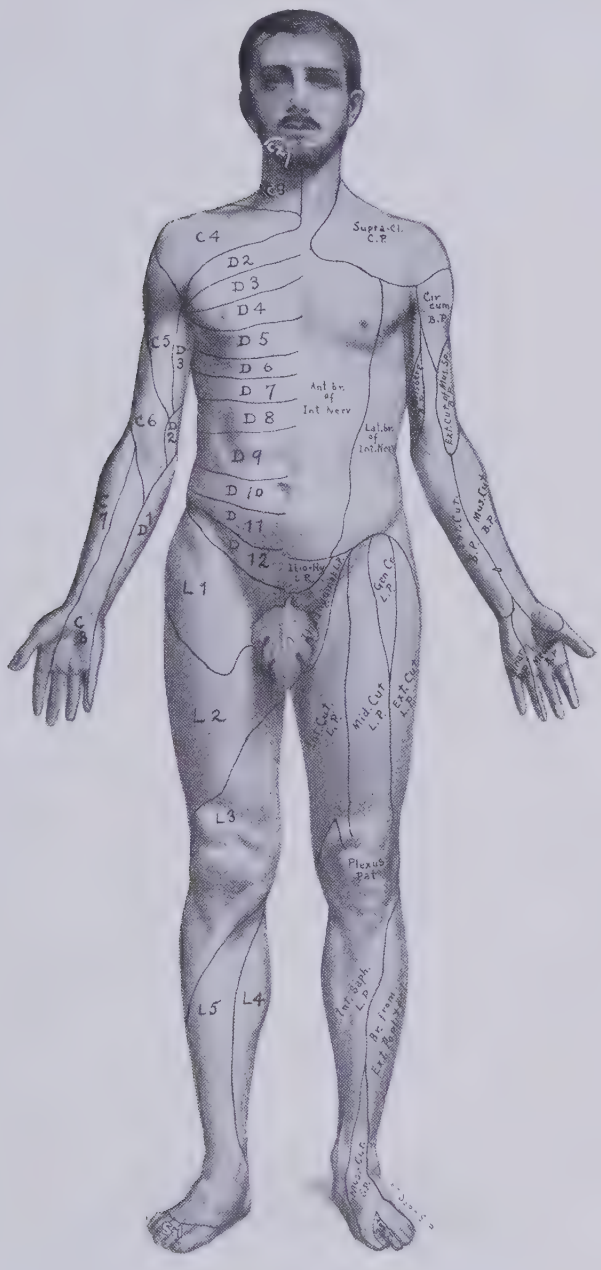


FIG. 26.—Showing the segmental or root distribution of cutaneous sensory nerves on the right side, and the peripheral distribution on the left side. (Drawn by H. T. Shannon.)

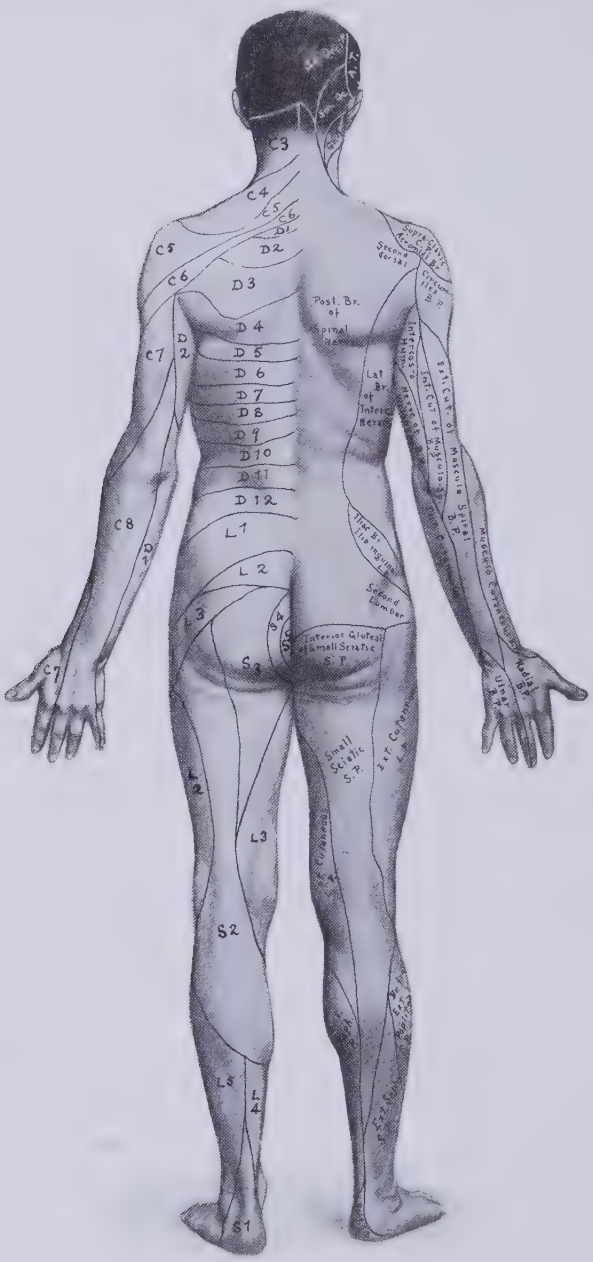


FIG. 27.—Showing the segmental or root distribution of cutaneous sensory nerves on the left side, and the peripheral distribution on the right side. (Drawn by H. T. Shannon.)

or cold sometimes exists. These conditions are called hyperthermalgesia and hypercyalgesia (Skinner, Starr). When the heat or cold is intense, a sensation of pain is felt. Cold pain is produced more easily in some places, such as the elbow, than in others, as for example, in the finger tips. Cold pain is produced by temperatures of from + 2.8°C. to − 11.4°C. Heat pain is produced by temperatures of from 36.3°C. to 52.6°C.

The *pain sense* is tested by pricking the skin with needles or the sharp points of an æsthesiometer. The faradic battery with metal points or a wire brush may also be used. Instruments for pinching the skin and measuring the sensibility by the strength of the pinch have been devised. The power of localizing pain is lessened in proportion to the analgesia. Pain and temperature sense are usually affected together.

The *pain sensation of muscles*, is tested by passing the faradic current through the part or by deep hard pressure into the muscle.

The epicritic and protopathic sensibility as described under symptoms are tested practically by cotton-wool giving light-contact sense and

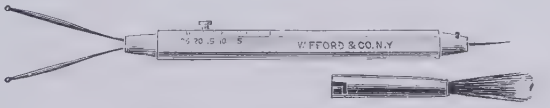


Fig. 28.—Author's æsthesiometer.

pin pricks giving sharp pain sense. The tests give an indication of the degree and location of peripheral nerve injury (Head).

The *vibration sense* is tested by placing a low-pitched tuning-fork upon the bony prominences. The feeling of vibration thus communicated varies about as does the tactile sense, but it is said to be lost in diseases in which the spinal roots are involved, such as tabes and in serious transverse lesions of the cord.

The tactile sense, as well as the other special and the general sensations, may show a delay in conduction. The tactile and pain sense especially should be tested on this point. The delay may amount to several seconds. It is especially characeristic of tabes dorsalis.

The *stereognostic sense* is tested by placing variously shaped objects in the patient's hands and asking him to name them. It depends upon the integrity of the tactile or of the deep, *i.e.*, musculo-articular, sensibility or upon that of the stereognostic centre in the parietal lobe.

The normal nerve-supply of the skin is shown in Figs. 26 and 27.

Tests for Deep Sensibility.—Anæsthesia of the special sensory nerves of the muscles, joints and tendons causes ataxia and inco-ordination. Muscle anæsthesia causes chiefly a loss of *weight sense* or loss of power to determine weights. It is tested by the use of weights suspended by a

string so as to exclude pressure sense; also by causing the patient to squeeze a dynamometer up to a certain fixed number.

In articular and tendinous anæsthesia there is loss of posture sense. It is tested by the physician's moving the patient's limbs and having the blindfolded patient tell in what direction the movement is made. Or he is told to follow with one limb the movements which the examiner makes with the other, or the patient shuts his eyes and tries to put the tip of his finger to the tip of his nose (finger-nose test) or the heel of one foot on the patella of the other leg (heel-knee test).

Muscular, articular and tendinous anæsthesia usually exist together; Such ataxia shows itself in standing and in locomotion and other voluntary movements. Thus we have a *static* ataxia and *locomotor* or motor ataxia. Static ataxia, or inability to stand (or sit) without swaying or irregular movements, is tested by making the patient stand with the eyes closed and the heels and toes close together. Normally, the head moves not over an inch in this position, and the patient holds the head and body more rigid with the eyes closed than with them opened. In ataxic states the reverse is true, and decided swaying or even complete loss of equilibrium occurs with the eyes closed, or even with the eyes open, and the base narrowed by putting the feet together. This phenomenon is called the "Brauch-Romberg symptom." In static ataxia, muscular and articular sensations are both involved.

Ataxia of motion is tested by observing the gait and the movements of the extremities. The patient cannot walk a straight line and cannot walk without watching the floor with the eyes. The arms cannot be moved in a co-ordinate way. With the eyes closed, the patient cannot place the finger on the tip of the nose or lobe of the ear or any indicated spot. Ataxia of motion involves especially the articular and tendinous sensations, but not these exclusively. It may be measured by noting how close in walking the patient keeps upon a given line 10 feet long; how near he can place the finger upon a centre of a board marked like a target. The patient is placed 10 feet away and made to walk directly at it and place the finger in the centre.

Hypotonia or loss of muscular tonus is tested by having the patient lie on a couch. The physician takes the foot and lifts the leg by it, the knee being kept stiff. Ordinarily the leg cannot be carried quite to a right angle without the knee bending. If it does go to a right angle or beyond, there is hypotonia of the limb. The fingers, wrists, toes, ankles, knees show also abnormal flexibility. This condition is natural to some extent in children and it is sometimes a physiological characteristic of the adult.

Cerebellar and Vestibular ataxia or asynergy.—Ataxia of gait and station may be caused by lesions of the cerebellum and its connecting paths,

and of the vestibular nerve. These impair the apparatus of equilibrium and cause uncertain, jerky, staggering movements resembling somewhat the ataxia above described. The co-ordination of the arms is also involved. The tests and description of cerebellar and vestibular asynergy will be given under the description of cerebellar disease.

Besides the foregoing methods of examination and testing for nervous conditions, there are a number of epinomic tests which are of importance in investigating certain special neuroses. I have put the description of these tests in connection with the disease to which they are related; but the following list of the tests collated for me by Dr. H. W. Frinck is given here as a matter of convenience in reference:

Abadie's Sign (of Tabes)
Babinski's Test (of Chronic Paralysis)
Barany's Test (of the Labyrinthine Reflex)
Biernacki's Test (of Tabes)
Brissaud's Test (of Organic Paralysis)
Brudzinski's Neck Sign (of Meningitis)
Chvostek's Sign (of Tetany)
Erb's Sign (of Tetany)
Grasset's Test (of Hemiplegia),
Hoover's Sign of (Hemiplegia)
Kernig's Sign (of Meningitis)
Mannkopf's Sign (of Simulated Tenderness)
McEwen's Sign (of Hydrocephalus)
Mendel-Bechterew Test (of Py. Tract lesion)
Moebius's Sign (of Exophthalmic Goitre)
Quinquard's Sign (of Chronic Alcoholism)
Rinne's Test (of Hearing)
Romberg's Sign (of Ataxia)
Rosenbach's Sign (of Hemiplegia)
Schwabach's Test (of Nerve Deafness)
Stellwag's Sign (of Exophthalmic Goitre)
Trousseau's Sign (of Tetany)
Von Graefe's Sign (of Exophthalmic Goitre)
Weber's Test (of Hearing)
Wernicke's Hemiopic Pupillary Reaction

Sense of Vision, Hearing, Vestibular Sense, Smell and of Taste.—See Cranial Nerves.

Examinations of the *X-ray* are useful in determining the presence of fractures, bony growths, atrophies and deformities. Occasionally the X-ray photograph helps in the recognition of brain tumors.

Blood examinations are necessary in acute inflammatory conditions, syphilis, pernicious anæmia, etc. It is hardly necessary to speak of the need of tests of the urine or of those which inform us regarding conditions in the gastro-intestinal tract. In many forms of nervous diseases every organ and fluid of the body must be examined so that the laboratory is an especially important aid to neurological diagnosis.

Examination of the Cerebrospinal Fluid.—The cerebrospinal fluid is normally a clear liquid with a specific gravity of 1006 to 1008. It is alkaline in reaction and contains a trace of serum-globulin and a substance which reduces Fehling's solution. There may be one or two lymphoid and epithelial cells. In certain nervous diseases, such as tabes, spinal syphilis, paresis and in inflammatory diseases, various changes are found. The special characteristics are referred to under the different diseases.

The technic of spinal puncture as illustrated and described for me by Dr. MacRoberts of the New York Neurological Institute is as follows: The cerebrospinal fluid can be obtained in a safe and simple manner by introducing a long hollow needle equipped with a stylet through the dura mater in the lumbar region of the spine. The needle is specially constructed for this purpose. The Hastings needle, the one generally used, is of strong flexible steel of fine bore with a handle large enough to allow of firm grasp.

The needle should be boiled and the operation carried out under sterile precautions.

The patient is best seated on a chair or the edge of his bed with his back rounded toward the operator. If weak or unable to sit up he may

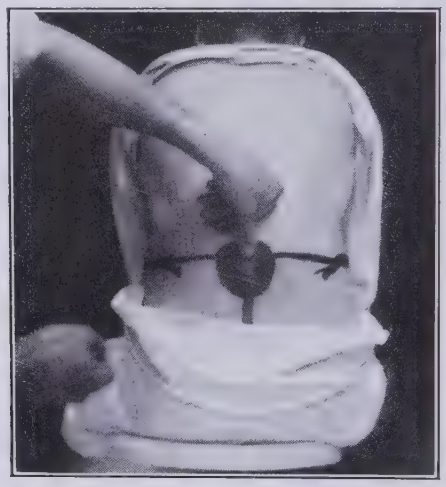


Fig. 29.—Spinal puncture. The needle is inserted along the lower edge of a spinous process marked by the tip of the thumb.

lie on his side with knees drawn up toward his chest. In cases of brain tumor or of greatly increased intracranial pressure the patient should be on his side. In cases of posterior fossa tumor or marked distention of the ventricles the puncture can be performed with comparative safety if the foot of the bed be first elevated with shock blocks and allowed to remain in this position for eight hours or more after the withdrawal of the fluid.

It has been found advisable to tell the patient in a few simple words what is about to be done, saying that he may expect the pain of a pin prick as the needle goes through the skin, but if he then keep very still the remainder of the procedure can be completed in a few moments without causing him further pain.

The point of election for the insertion of the needle is between the third and fourth or fourth and fifth lumbar vertebræ, in the median line or slightly to one side. The location is best obtained by making, with an applicator dipped in tincture of iodine, a straight line which unites the iliac crests. Where this line crosses the line of the spinous processes a space between the spines will be found by palpation with the thumb. The skin about this point should be sterilized with alcohol and tincture of iodine. The thumb of the left hand should be placed on the spinous process above the interspinous space with the edge of the thumb even with the lower edge of the bone. The needle should be grasped firmly

Fig. 30.—Spinal puncture. Showing the position of the patient and the position of operator's thumb palpating the lower edge of a spinous process.

with the base against the palm and the shaft steadied with the fingers, and the point placed just below the thumb edge and thrust smartly through the skin. It may then be more slowly pushed through the ligamentous structures until by the change in resistance it can be felt to have pierced the dura mater. The stylet should then be withdrawn and 4 or 5 cc. of the fluid collected in a sterile test-tube. The needle is then carefully but quickly removed and a small square of gauze fastened over the puncture point with adhesive.

The fluid should be corked and placed in the ice box and should be examined some time within the next twenty-four hours.

Following the puncture it has been the custom to have the patient lie flat on his back for twenty-four hours. It has been found recently that, by allowing our patients up sooner, fewer headaches resulted.

The fluid is later tested for blood, excess of globulin, the Wassermann reaction, the number and character of cells, cholin, sugar, and bacteria. The special methods and indications are given under the description of meningitis, syphilis, cerebral hemorrhage and cerebral abscess, and cord tumors.

CHAPTER VI

HYGIENE, PROPHYLAXIS, TREATMENT

In the treatment of nervous disease, the physician attempts to relieve distressing symptoms, to secure radical cure, and to prevent return. This calls for various measures which may be classed under the heads of general hygiene, diet, exercise, climate, hydrotherapy, massage in various forms, electricity, drugs, external applications and surgical intervention.

General Hygiene.—To secure and keep steady nerves and to prevent the supervention of organic nervous disease, would require a considerable reconstruction of the present social system. The struggle for existence and success is keener than it used to be. There is a smaller field for the slightly backward class, and more men fail in life and become parasitical than formerly when there was a wider field for the simpler manual occupations.

I can only give some hints as to the kind of advice physicians should give to help along those who are very likely to be handicapped by a neuropathic constitution. Thus two people of very nervous temperament or bad stock should not marry. Blood relations of the same temperament should not marry, and families with a psychopathic taint should not intermarry. Children should be brought up to eat slowly a mixed diet, to sleep early and long, to play in the open air, to learn self-control and obedience. Their parents should keep from them all infective fevers. Systematic study and work are good for all children. It is the strain due to defective vision, poor light and ventilation and unsuitable tasks that hurts the neurotic. Education and occupation are the best kind of builders up of healthy nerves. There are children, however, who cannot follow the ordinary educational lines and who must be specially trained in consequence. The queer and eccentric children with some twist, or precocious talent, need especial care. They usually must be brought up to follow lives on a low mental plane. Too many good farmers and artisans are spoiled by being made poor professional men, or being set up in responsible business positions. Adults need to keep in mind especially: moderation, exercise and the avoidance of a luetic infection. With these most need not fear the use of alcohol, tobacco, tea, coffee, or even occasional irregularities in sleeping and eating. Physical and mental strains, infective fevers and toxins are prolific pro-

moters of nervous disease. Syphilis stands out as the most important single factor in producing organic nervous diseases. If it could be removed we would have no locomotor ataxia, or paresis, less myelitis, and far fewer cases of apoplexy.

Alcohol is a less important factor, but does much to produce mental disease, vascular disease, and hereditary degeneration.

Diet.—There is no definite formula for diet in nervous diseases. The kinds of foods which are suitable for infants and children, and growing youths, are not equally suitable for the mature or the senile. There are some general facts, however, which may be laid down, regarding the diet for the nervous; that is to say, for persons who have a more or less neurotic and unstable constitution. This class of persons seems to be, as a rule, unable to tolerate sweets or large amounts of flesh food, and they do best upon a mixed diet which contains a moderate amount of proteid together with vegetables and fruit. The question, whether neurotic patients do better upon the so-called "high" proteid of flesh or the "low" proteid of vegetables, does not seem yet to be settled, though the practice is, on the whole, rather more in favor of using the flesh proteid than the vegetable proteid, especially in the early half of life.

There are four kinds of what may be called general diets prescribed for patients. I do not mean to include in these four, however, the special diets that are used in the acute and organic diseases, such as diabetes and fevers, serious gastric disturbances, and so on.

These four diets are: first, vegetarianism; second, the modified vegetarian or purin-free diet; third, the Salisbury high-proteid diet, and, fourth, the Chittenden or "low diet."

The pure vegetarian diet is one which has no scientific basis, and which seems to me, on the whole, as unwise, though it apparently suits certain constitutions, and is, if anywhere, especially suitable for those of rather advanced years.

The purin-free diet is vegetarianism plus the use of milk and cheese. In this, there is a reduction of the proteids, but an increase somewhat, of the fats and carbohydrates.

The Salisbury diet consists of lean beef and hot water, and its use involves a large increase of the proteids and of the purin-bodies, while there is a decrease of the fats and carbohydrates. This diet is usually somewhat modified by the addition of bread or toast, and sometimes of fruits, so that the diminution in the carbohydrates is not so marked.

The Chittenden diet is one in which the food, as a whole, is cut down in all its forms, the proteid being reduced nearly one-half. Experience has shown, that all these different diets may lead to the same therapeutic results—that is to say, that a purin-free diet will cure a mi-

graine or lessen the attacks, and that a Salisbury or meat diet may do the same.

The injury caused by any form of diet is largely due to the fact that putrefactive changes occur in it, leading to toxæmias, or that there gets into the blood an excess of food or fuel; and if this diet is modified so that such an excess is lessened, without weakening the system, we can secure good results. In fine, the main factor in prescribing any diet, is to see that it is a digestive and assimilable one, and that there does not get into the system an excess of fuel, producing what is termed a "hyperpyræmia."

In elderly persons the amount of nitrogenous food needed in the form of meat is usually less; and they often do well on a vegetarian diet. As a rule, however, vegetarians after a few years find that a return to some carnivorous food is indicated.

Some neurotic persons seem to need a great deal of food but, as a rule, harm comes from full diets and one cannot get strong by stuffing.

The frankly nervous, the hypomanic and psychasthenic patients should not use alcohol at all. Tea and coffee can be taken in very small amount and best without sugar. The various alkaline mineral waters may be used temperately with impunity, but none of them have much specific effect in relieving nervousness or curing the nervous temperament.

Exercise.—As a prophylactic against nervous disorders, the value of exercise, especially if taken out of doors, can hardly be overestimated. Brain workers are better for moderate exercise, but they do not need much; and after twenty-five, severe intellectual work can rarely be done by persons in athletic training. Before the age of twenty-five, when the system is exuberant with vitality, hard study and hard physical exercise can be pursued successfully together by some. Persons of a neuropathic constitution are most benefited by regular exercise when it interests the mind. Indoor gymnasium exercise with the ordinary apparatus does little good except through the bath that follows it. In many forms of chronic organic nervous disease, exercise is to be prohibited.

The most important line of effort in prophylaxis is the warding off of excessive or premature arteriosclerosis. The surest preventive of this is a good heredity. But, given an average constitution, one can delay arterial degeneration only by leading a perfect life, that is to say, the individual must avoid all mental and physical strain and shock, alcohol, excesses in eating and tobacco. He must not get syphilis. He must keep the emunctories open and get as much as possible of fresh air. All of which is the council of perfection, except for children. Here wise parents can do much to prevent the development of arterio-sclerotic neuroses.

Hydrotherapy.—Hydrotherapy is the science of applying water in the treatment of disease. The modes by which it is used in neurological therapeutics are:

I. General hydrotherapy:
   1. Tonic hydrotherapy.
   2. Sedative hydrotherapy.
   3. Indifferent baths for mechanical purposes.

II. Local hydrotherapy.

The details of the use of these measures are now embodied in special treatises and works on therapeutics.

Massage.—The term massage may be made to include all the manipulations of the body for the purpose of curing disease. The different methods of applying it as classified by Jacoby are:

Effleurage or gentle stroking. The maximum force to be applied here should not exceed the weight of the hand. *Massage á friction* or rubbing. Energetic strokes with one hand and strong circular or to-and-fro friction with the other. *Pétrissage* or kneading. *Tapotement* or percussion with the fingers, hands, or instruments. *Frictional movements*, passive, active and combined with movements are made by the operator. The physician may be reminded that a male operator is a *masseur*, a female a *masseuse*, and that the patient is *masséd*.

Massage accelerates the lymph and venous currents, and thus promotes absorption. It increases at least temporarily the number of red blood-cells (Mitchell). It increases the rapidity and force of the heart beat (except abdominal massage which slows the heart) and helps to relieve local congestions and inflammatory deposits. It presses and stretches the terminal nerve filaments, increases the irritability of motor nerves and the contractility of muscles. It may either increase or lessen the irritability of sensory nerves according as it is applied. Of the various forms of massage, *tapotement* is frequently useful and is the kind often used in neuralgias. It is applied not only with the fingers and hand, but also by the aid of rubber tubes known as muscle beaters, rubber balls with rattan or whalebone handles, percussion hammers and various percuteurs.

Massage is of considerable value in certain forms of atonic neurasthenia and hysteria associated with anæmia, dyspepsia and feeble circulation; in hemiplegia, in the paralyses of peripheral origin, in functional spasm, especially in some forms of writer's cramp and allied neuroses, in cerebral hyperæmia, insomnia, constipation and in headache and some neuralgias, especially those about the head, neck, and arm. It is contraindicated usually in heart disease, in advanced arteritis or when there is danger of dislodging a thrombus.

Regular educational muscular movements according to a certain fixed schedule are used in the treatment of locomotor ataxia and paralysis. The details of these exercises will be given later.

Osteopathy.—"A system of therapeutics based upon the theory that many diseases are due to pressure upon the vessels or nerves by some displaced vertebra or other part of the skeleton, or to a condition of imbalance of the muscles moving any joint; the treatment is directed to the mechanical correction, by means of manipulation, of the assumed osseous displacement or muscular imbalance, with the consequent repression of the abnormal reflexes and a restoration to normal of the circulation and the nerve impulses" (Stedman).

It is a form of treatment which gets results, according to my experience partly by suggestion and partly by improving mechanical conditions, as does massage. It is dangerous in certain forms of nervous disease like tabes. It is useless in others like Parkinson's disease. It is dangerous also, like all special therapeutic systems, when it makes large and unfounded claims.

Climate in Nervous Diseases.—The factors which make up a special kind of climate are: Purity of air; temperature; humidity; sunlight; rarefaction of air; ozone; wind; electricity; soil; trees, social conditions.

Regarding these points, some facts are very well settled. The air in the country is purer than in cities. The air on the sea and at high levels is purer than in other localities. The temperature above the sea level diminishes about 1°F. for every 300 to 350 feet, and is less the dryer the air. Alterations in temperature are less near the sea and less in the southern hemisphere. The higher the elevation and the colder the air, the less moisture does it contain. About the factors of ozone and electricity in the air little definite is known. As to sunlight, an excessive amount of it is said eventually to enervate patients.

Climates are classified by Weber into marine, low-level inland and high-level inland. These all have great variations in quality, depending upon their temperature, moisture, etc. As a general rule, warm marine climates and sea voyages are best for neurasthenic invalids of the irritable type. On the other hand, in atonic and anæmic conditions high inland climates are better, at least for a time. Such climates should not be too dry or windy.

In organic degenerative diseases of the nervous system, marine climates and low levels are better. As a whole, it seems to be the conclusion that plenty of fresh air is the essential in all climates and is better than any special climates. Relatively short changes from low to high levels or from temperate to tropic climates often leave a distinctly tonic effect.

Germany, the Riviera, the Bermudas, the Azores, the West Indies, southern Colorado, Arizona and southern California are favorite places for sending neurasthenic Americans. Camp life in the Adirondacks or other forests is also found most useful.

ELECTRICITY IN NERVOUS DISEASES

Technical Terms.—There are certain technical terms which it is necessary to understand. *Electromotive force* (symbol, EMF) is the force which tends to set electricity in motion. An electric current results. The *current strength* (symbol, C) is the term used to express the capacity of the separated fluids to overcome resistance in their attempts to reach equilibrium or equalization again. This current strength, or simply the current, naturally is in proportion to the strength of the electromotive force, which is constantly dissociating the electrical fluids and generating the current. If, however, as is always the case, the electrical fluid meets resistance in seeking equilibrium, the resistance diminishes its current. Hence we have the formula known as Ohm's law:

$$\text{Current strength} = \frac{\text{Electromotive force}}{\text{Resistance}}; \text{ or } C = \frac{EMF.}{R}$$

All bodies offer some resistance to electrical currents, and it is important to have some standard unit of resistance for the sake of comparison. Such standard unit has been adopted and is called an *ohm*. It is the resistance offered to a current by a certain piece of wire of definite size and length.

A *volt* is the unit of electromotive force, *i.e.*, it represents the force which will generate a certain amount of electricity in a second of time. A Daniell cell is not quite one volt strength.

An *ampere* is the unit of working power or current strength. It is the current strength produced by one volt of electromotive force working against one ohm of resistance. A milliampere is one-thousandth of an ampere. A *watt* is the unit of work.

When a given current flows along from a large into a small conductor, the quantity in this latter conductor in a given section is greater and the current is said to be denser. The instrument by which the strength of a current is measured is known as the amperemeter; in medical practice, only fractions of the ampere are used, and the instrument is called the *milliamperemeter*. A *rheostat* is an instrument for interposing resistance in a current.

Electrical Appliances.—The batteries used in neurological practice are: the static, the faradic, the galvanic, and the high frequency.

The current of the faradic battery varies in quality in accordance, (1) with the length and number of turns in the coil; (2) the form of the electrical wave; (3) the number of interruptions; and, (4) the strength of the battery.

The currents from short coils of coarse wire have a lower potential and slightly more voltage. They are more efficient in producing mus-

cular contractions and are more irritating to the sensory nerves. These qualities are increased with slow interruptions of three or four per second. When muscular contractions and mechanical exercise with stimulation are desired, the short coils (primary or secondary) with slow interruptions are indicated. The current from long coils has a higher potential and less voltage; it has less power in contracting muscles and a different effect on the sensory nerves. When the interruptions are very rapid and the coil is very long, the effect on the sensory nerves seems more sedative; perhaps, in part, because of a change in the form of the electrical waves.

The physiological effect of a current depends, as I have stated, in part upon the character of the wave of electrical force. If this is high and sharp, the stimulation is different from that produced by

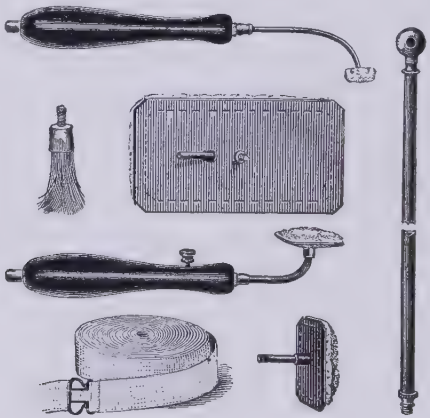


Fig. 31.—Author's electrode set.

a wave which gradually rises to its height. D'Arsonval has devised an instrument for producing these blunt-topped waves, and at the same time reversing the current. The current is called *sinusoidal*. I am not aware of any special therapeutic results in neurology from this kind of current, though it is claimed that very slow wave-like currents affect better involuntary muscles. The Leduc current is a rapidly interrupted galvanic current, the period of duration of the current being one-tenth that of the intermission. It is claimed that when sufficiently powerful, 20 to 60 volts or more, it produces local anæsthesia and when passed through the head to produce general anæsthesia and sleep.

Very rapidly interrupted and alternated currents of extremely high voltage are called high frequency currents. When passed through the entire body they lower blood pressure 5 to 10 points, and increase metabolism. When applied locally under the name of "violet-ray" curents they have an efficient counterirritant effect.

Galvanic Batteries.—The ordinary accessories to the faradic and galvanic batteries are electrodes, rheostat and milliamperemeter.

The electrodes needed for ordinary purposes are: An indifferent electrode measuring 5 cm. by 15 cm. A normal electrode, 10 sq. cm. A unit electrode, 1 sq. cm. A soft wire brush. Three handles: one 10 cm. and one 40 cm. long, one short handle with an interrupter. A milliamperemeter. A rheostat. (See Fig. 33.)

Methods of Application.—Static electricity is applied for fifteen or twenty minutes daily or tri-weekly. For general tonic or sedative effects, sparks are drawn from all parts of the body except the face. In paralysis or spasm or pain, sparks are applied to the affected area. For headaches and cerebral paræsthesiæ the electrical breeze is very useful, but it must be strong.

The faradic and galvanic currents are used for about the same time and intervals as the static. In some cases, however, the galvanic current should be given daily or even two or three times a day. As a rule, a course of electrical treatment should be continued for six to eight weeks, and then discontinued for a time.

In general electrization, whether galvanic or faradic, the indifferent electrode is placed on the sternum, feet or back, and the other pole is carried over the limbs, trunk, neck and, if indicated, the head. In some cases, however, the two electrodes are applied together upon the different muscles of the body. In local electrization, the large electrode may be applied on an indifferent spot and the other applied to the affected limb or limbs, or the two electrodes may be used together on the same segment of muscles. There are special points at which the muscular contraction is most easily brought out. These are called the motor points.

Electro-diagnosis.—When a motor nerve is cut off from its centre in the spinal cord, or when this centre itself is diseased, the nerve and later the muscle undergo a degeneration. As a result of this, their reaction to electrical currents is changed, and we get what is termed "partial degeneration reactions" and "complete degeneration reactions," according to the degree of disturbance. These reactions are due mainly, if not wholly, to the degeneration in the terminal nerve-fibres and motor end-plates in the muscle. When the muscle alone is diseased, the reaction is not changed until very late. The change in irritability is due to the fact that as the nerve-fibre wastes it takes an electric current of comparatively long duration and considerable strength to stimulate it.

The first effect is to cause it to lose its contractility or reaction to weak currents, then to extremely rapid, short currents like the static, then to the faradic, and last to the galvanic. Such change is known as the *quantitative alteration* in electric irritability.

But besides this, the nerve and muscle are affected in a different way by the different poles of the galvanic battery. In normal nerve and muscle, a contraction is caused more readily by the negative pole than by the positive. But muscles with degenerated nerve supply sometimes respond as well or better to the positive pole. This forms what is called the *qualitative* or *serial change* in the irritability of the muscle.

Finally, degenerated muscles respond more sluggishly than normal to the galvanic and faradic currents. The contraction, instead of being sharp and jerky, is sluggish and almost tetanic. This is called the *modal change* in irritability, and it is far the most important sign of muscular degeneration.

The *qualitative* change is gotten only by placing the active electrode over the muscle, but the *quantitative* and *modal* changes may be gotten by placing the electrode over the nerve as well as over the muscle. In describing these changes, the following abbreviations are used:

DeR = degeneration reaction.

AnCC = anode or positive-pole closure contraction.

CaCC = cathode or negative-pole closure contraction.

AnOC = anode opening contraction.

CaOC = cathode opening contraction.

Te = tetanus.

D = circuit is closed and current flowing.

AnDTe = tetanic contraction while the positive pole is applied and the circuit closed.

The sign > means greater than; <, less than. Thus AnCC < CaCC means anode closure contraction is greater than cathode closure contraction.

Degenerations in nerve do not occur except in lesions of the peripheral motor neuron and in very late stages of primary atrophy of muscles. Hence, when one finds degenerative reactions he can almost absolutely exclude disease of the brain, functional disease and primary disease of the muscle. The following rules may be formulated for testing for degeneration reactions:

Use the faradic current first.

The Faradic Current.—Use a secondary induction coil of wire 0.225 mm. in diameter and 800 m. long. The distance over which the coil moves is divided into a hundred parts. The strength of current is indicated by percentage or millimetres.

Record the minimum necessary for muscular contraction, using the same electrodes and in the same way as in testing with galvanism.

The Galvanic Current.—(1) Place the indifferent pole over the sternum and a 10 sq. cm. electrode over the muscle. (2) Pass the current for one minute. (3) Then find the minimum strength needed for a cathode closure contraction. (4) Then for an anode closure contraction. Repeat this test three times. (5) With a given cur-

rent, note whether the cathode closure contraction is stronger than AnCC or otherwise. Test this three times. (6) Note the character of the contraction, if sharp or sluggish. (7) Test nerve in same way.

The qualitative changes may be expressed by a formula like AnCC = or > CaCC; *i.e.*, the positive-pole closure contraction is equal to or greater than the negative-pole closure contraction. Or, better, the minimum strength of current required to cause a contraction in the muscle is recorded for the positive pole and for the negative. Thus:

AnCC 5 ma. or 8 cells.
CaCC 4 ma. or 6 cells.

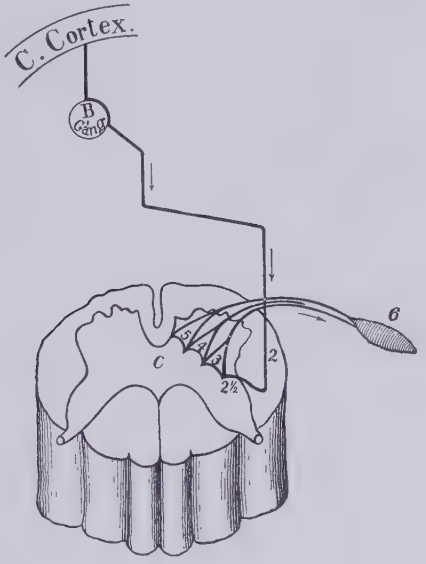


FIG. 32.

The following table and diagram (Fig. 32) show the diseases in which degeneration reactions may be expected:

TABLE SHOWING THE LESION, ITS RESULTS, THE NAMES OF THE DISEASES AND THE ELECTRICAL REACTIONS

Lesion of—	Result	Disease	Electrical Reaction as to Qualitative
1 to 2½. Cortex to cord.	Paralysis, contractures.	Hemiplegia from hemorrhage. Embolism, tumors, lateral sclerosis.	Nerve: normal. Muscle: normal.
3, 4 and 5. Cornua.	Paralysis, degenerative atrophy of nerve and muscle.	Acute and chronic anterior poliomyelitis.	Nerve: DeR. Muscle: DeR.
2 to 5	Paralysis, contractures.	Amyotrophic lateral sclerosis.	Nerve: normal Muscle: normal or partial DeR. When disease is advanced.
5 to 6.	Degenerative atrophy, of muscle. Later, degeneration of nerve.	Progressive muscular atrophy, bulbar paralysis.	

TABLE SHOWING THE LESION, ITS RESULTS, THE NAMES OF THE DISEASES AND THE ELECTRIC REACTIONS.—(*Continued*)

Lesion of—	Result	Disease	Electrical Reaction as to Qualitative
Nerve.....	Paralysis, degenerative atrophy of nerve and muscle.	Neuritis, from wounds, toxemia or pressure.	Muscle: DeR. Nerve: DeR.
Muscle.....	Wasting, paresis.	Simple atrophy; primary or idiopathic myositis. Pseudomuscular hypertrophy; other types of primary myopathies. Rheumatic atrophy and paresis.	Nerve and muscle normal until late in the disease.

It should be said, finally, that it is the *sluggish contraction* which is the most important element in showing degeneration; also that it is the muscle which should be tested most carefully, as only over it does one get the qualitative changes.

Therapeutics.—Electricity is used as a counter-irritant and as a general mechanical tonic in states of muscular and nervous weakness. It is used in paralysis, spasm and pain, and for its supposed specific action in certain functional and organic diseases.

The faradic and static currents have a counter-irritating, stimulating and excito-reflex effect. The galvanic current has a sedative and antispasmodic effect. The high-frequency current promotes metabolism and temporarily reduces blood-pressure.

The "violet-ray" current acts mainly as a counter-irritant.

Electrolytic, cauterizing, and cataphoric effects are also produced, but are rarely needed by the neurologist. A considerable portion of the effects of electricity are psychical, but they are not the less real or valuable.

Radiant Energy in the form of X-rays, ultra-violet rays, radium emanations have been of more help to surgery than medicine in therapeutics. The X-ray and radium are said to have some effect in neuritis and neuralgia, but the results are not uniform or as yet conclusive.

The X-ray with the Coolidge tube and radium have some effect in destroying new-growths, and are being used in spinal-cord and cerebral tumors.

PSYCHOTHERAPY

Psychotherapy is a term applied to that form of healing in which mental influences are used to correct morbid processes.

Psychotherapeutics has always been used in an irregular and casual way to cure disease. Modern therapeutics endeavors to formulate definite rules for its use and to indicate systematically the conditions for which it should be applied.

Hypnotism is the oldest and best known method. In hypnotic therapeutics the patient is put into a partial or complete hypnosis. Suggestions and directions as to cure are then made, which the patient on coming out of his hypnosis follows. This method of treatment is uncertain and difficult and is of use only in a narrow field. It has been practically abandoned, though occasionally its use gives brilliant results, mainly in hysterical conditions, morbid habits of not long standing, and in the minor psychoses. It is more effective in the younger subjects. It is of no use in the serious psychoses. (See Hypnotism.)

Suggestion with minor hypnosis is only a modification of the preceding method. Here the patient is simply persuaded into a quiet, receptive mood or into a slight degree of hypnotic trance, which is oftener really only a slightly somnolent state. Then suggestions are patiently and insistently repeated. This method is more simple and on the whole more useful than the first. It has to be repeated perhaps as often as a dozen times before the desired impression is secured. But it is fairly efficient in the pychasthenic, more particularly those with fixed ideas, morbid fears, attention pains and morbid melancholic moods. The treatment is most useful here also in cases that are not of very long duration. It deserves a permanent place in neurological therapeutics, but it can only be applied by those who have a thorough knowledge of the patient, of the disease and, I may add, of themselves. It requires a steady and forceful and confident personality. Formally and strictly applied, it has not a wide field. In a simpler and common-sense way, however, the method can be used by any physician, and is used, no doubt, all the time, for it really consists in giving the patient the vital sense of proportion in his judgment of things. In many patients the harmful thoughts are not entirely subconscious. The patient knows them, but has not quite recognized their importance and the need of treating them sanely and definitely and of putting them in their right relation.

The method of psychic analysis and reintegration. This is also called the method of Freud. It cannot be described in detail here. We assume that many of the morbid mental states and bodily perversions are dependent on the presence of a pernicious and perhaps long-forgotten subconscious activity.

Below the field of ordinary conscious processes there is memory of some earlier event acting, unknown to the patient, upon his conscious mental states and inducing in him certain morbid ideas or emotional moods which cause his sickness. The operator, by studying and questioning, by learning of his dreams, his "running associations," by telling him to empty himself of his troubles in a kind of "mental catharsis," brings into consciousness certain subconscious states which relate to events of his past. Perhaps also he finds that this lurking idea or

emotional state is doing mischief. He then confronts the patient with the facts, shows him what is the real cause of his obsession, persuades him of its unreasonableness or accustoms him to look upon it in a normal way. In other words, he brings the supposed subconscious cause of the trouble into consciousness, readjusts associations, and enables the patient to fight his trouble in the open.

It is further claimed by the psycho-analyst that the symptoms from which the patient is suffering, the spasm, the obsession, the morbid fear, are really the expression of a subconscious desire or of unsatisfied infantile instincts, usually of a sexual origin. My views as to the merits of this doctrine are given in the chapter on psycho-neuroses.

The Method of Re-education.—This may be called also the method of "therapeutic talk." It is known as the method of Du Bois. It is really an application of the principles of suggestion and of readjustment, or re-education, used in the preceding methods. The patient has his condition explained to him, the aims of his physician are placed before him, the reasonableness of his cure, the unreasonableness of his mental state and of his point of view are persistently argued over. The patient is sometimes persuaded, sometimes bullied into health. All this is done, however, after first isolating the patient from everyone and clearing the ground entirely of exogenous irritation. The doctor, with the nurse, a necessary adjunct, has the field entirely to himself. Obedience is enjoined. It is, in fact, a rest and isolation cure, combined with forceful presentations of the reasonableness and possibilities of cure. No medicines are given, as a rule. This is a method now having much vogue, but it is only a modification of the Weir Mitchell cure, in which the *psyche* receives rather more dominantly the attention of the physician.

In estimating the value of these cures it must be remembered that they are useful practically only in the psychasthenic and neurasthenic groups of patients. The mild recurrent melancholias so often mistaken for neurasthenia get well anyway. The melancholia of involution is little affected by these methods, nor are the major psychoses, if we except perhaps paranoia and early phases of such types of dementia præcox as are largely of psychogenic origin. The early and abortive types of the anxiety psychosis may also be affected.

Empirical Psychotherapy.—Therapeutic results are obtained by mandatory methods, by the method of extravagant promise or absolute assurance. These are the methods of the parent and guardian and of the charlatan and patent medicine label. Their success is more than counterbalanced by the evil done by inconsiderate assurance in improper cases, and by misleading persons to the use of measures not in the least indicated by their condition.

The employment of psychotherapy in conjunction with religious

work has recently been advocated and in some cities successfully carried out. A general indorsement of this union of medical and ecclesiastical work cannot be given, and its usefulness must have very definite limitations. Still, under very rigid control, it may prove an effective measure and be especially useful in counteracting the, on the whole, bad influence of various types and schools of "healers." One measure cannot cure all diseases or even all of one kind of disease, and the exploitation of infallible cures works an injury to society in the end.

Psychotherapy by Emotional Shock.—This is seen typically in the exhortations of religious healers and the dramatic methods of charlatans.

Philosophic Psychotherapy.—Most successful results are obtained among the always large number of suggestible and credulous minds by instillating a certain formula, squared by which their symptoms must cease and the disease disappear. This is the method of the Doctrinaire, or Philosopher, or Apostle. For example, it is asserted that God is good, God is all, therefore there can be no evil in you. Sickness is an evil, therefore it cannot exist. Therefore you cannot be sick. You are well. This doctrine ingeniously and eloquently poured into simple ears gradually gains conviction, the patient receives assurance, the attention is distracted from morbid symptoms and the patient gets well—sometimes. The doctrine applied to many thousands always reaches some who only need this distraction of attention from themselves. They ignore symptoms, and gradually their consciousness gets less sensitive to them. They harden their sensorium, and though they have conditions which would make some sensitive souls feel distressed, these conditions do not arouse their attention which is complacently and wisely directed elsewhere.

The only objection to this method of therapeutics is that its fundamental propositions are unproven, and its general application to surgery, infectious and organic disease is disastrous. But the philosophy of attention diversion to the healthy and holy side of life is wise and is indeed used by all physicians and in every form of scientific psychotherapeutics.

An essential thing in all forms of systematic psychotherapy is to have the patient on the proper stage. He or she must be removed from all influences but those of the doctor and his attendants. The atmosphere must be such that only his personality and views and counsels affect the patient. Then the work must be kept up for at least a few weeks and often longer. With this environment, the results are reasonably certain in proper cases. But without it, there is only a chance that the wisest counsel prevail.

Then the physician himself must be suited to the kind of work. He need not have a commanding personality, but he must be simple and impressive and sincere, and he must have no unpleasant personal habits.

The nurse, if there is one, must equally be a person of tact and experience, good personality and able to co-operate.

It can be seen that the systematic application of psychotherapeutics, at its best, is not possible for all, and is not suitable for most. Its unsystematic or rather informal use will always be a part of the equipment of the wise physician. It makes it all the more imperative that physicians be good as well as wise.

CHAPTER VII

DISEASES OF THE PERIPHERAL NERVES

Introduction.—The peripheral nervous system consists (1) of twelve pairs of cranial and thirty-one pairs of spinal nerves, with their root ganglia and terminal sense organs; (2) and of the sympathetic nervous system including the autonomic portion of Langley. The peripheral nervous system, therefore, to use the language of modern anatomy, is composed of peripheral motor neurons, peripheral sensory neurons, and peripheral ganglionic neurons (Minot).

GENERAL PATHOLOGY

Hyperæmia and Anæmia.—Under the head of hyperæmia and anæmia there occur types of nerve irritation, leading to different forms of neuralgia, paræsthesia and motor weakness or irritation. Hyperæmia

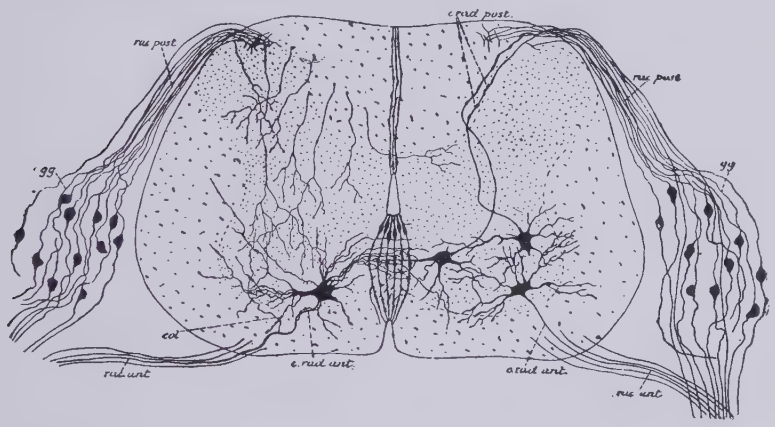


Fig. 33.—Showing the cells of origin of the motor nerves in the anterior horns of the spinal cord, and the cells of origin of the sensory nerves in the posterior spinal ganglia. (*Van Gehuchten.*)

and anæmia are, however, secondary conditions and are rarely recognized clinically. It cannot always be determined whether an irritated nerve is congested or anæmic, or whether the central part of the nervous system is not mainly at fault.

Angina of the Nerves.—In arterio-sclerotic conditions of the blood-vessels of the posterior ganglia and nerves there occur spasmodic closures

of the vessels or, in other words, anginal attacks, which cause severe neuralgic pains. The pathology of this condition will be referred to later.

Inflammation of Nerves—Neuritis.—There are two principal forms of neuritis: (1) Interstitial and perineuritis. (2) Diffuse neuritis with parenchymatous degeneration or parenchymatous neuritis. The two forms may be acute or chronic.

In the first type there is hyperæmia, with sometimes extravasation of blood. An exudation occurs into the fibrous framework of the nerve, with migration of leukocytes. The inflammation may become suppurative or gangrenous. If severe, it destroys the nerve-fibres; but oftenest the axis-cylinders are not destroyed, and recovery takes place.

In mild grades of perineuritis, such as occurs in sciatica, there is perineuritic exudation, a kind of serous inflammation with not much cellular proliferation. This perineuritis is probably the main factor in the typical neuralgias like sciatica neuralgia.

Chronic interstitial neuritis and perineuritis are accompanied with hyperplasia of the connective tissue, compression and more or less destruction of the nerve (Fig. 34). It may ascend or descend, and it is called, accordingly, *ascending*, *descending*, or *migrating* neuritis. It may affect only certain segments of the nerve, when it is called segmental neuritis or disseminated neuritis. Tuberculous and syphilitic neuritis are of the chronic interstitial or diffuse type. These latter forms rarely involve peripheral nerves, but rather the intracranial parts of the cranial nerves and the spinal nerve-roots in meningeal tuberculosis or syphilis. A syphilitic peripheral multiple neuritis is, however, thought to occur sometimes. Leprous neuritis is a very typical form of proliferating chronic perineuritis. Cancerous neuritis sometimes occurs and it is of the diffuse type, though sometimes an actual cancerous process invades the nerve.

The second type is called parenchymatous or degenerative neuritis and this process of degeneration is the dominant one, so that the changes can be best described under the head of degeneration of nerves.

Degeneration of Nerves.—This is a process in which the nerve-fibres gradually die; the myelin sheath and axis-cylinder disappear, leaving only a strand of connective tissue.

There are three forms of nerve degeneration: (1) primary; (2) secondary; (3) neuritic or toxic.

1. The primary form is rare, slight in extent, and of little clinical significance. In it there is simply a gradual wasting and disappearance of the axis-cylinder and myelin sheath. It occurs in old age, wasting diseases and as part of locomotor ataxia.

2. Secondary degeneration or Wallerian degeneration. This form

occurs when the nerve is cut across or compressed, or destroyed by inflammation, neoplasms or injuries.

The essential part of the nerve-fibre, the axis-cylinder, is simply a prolongation of the process of a nerve-cell. Its next essential part is the myelin sheath. This is of epiblastic origin and consists of a hollow cylinder inclosed in a thin membrane and containing a fatty substance. In degenerative processes of peripheral nerves the medullary sheath is first affected, then the axis-cylinder, least and last the neurilemma.

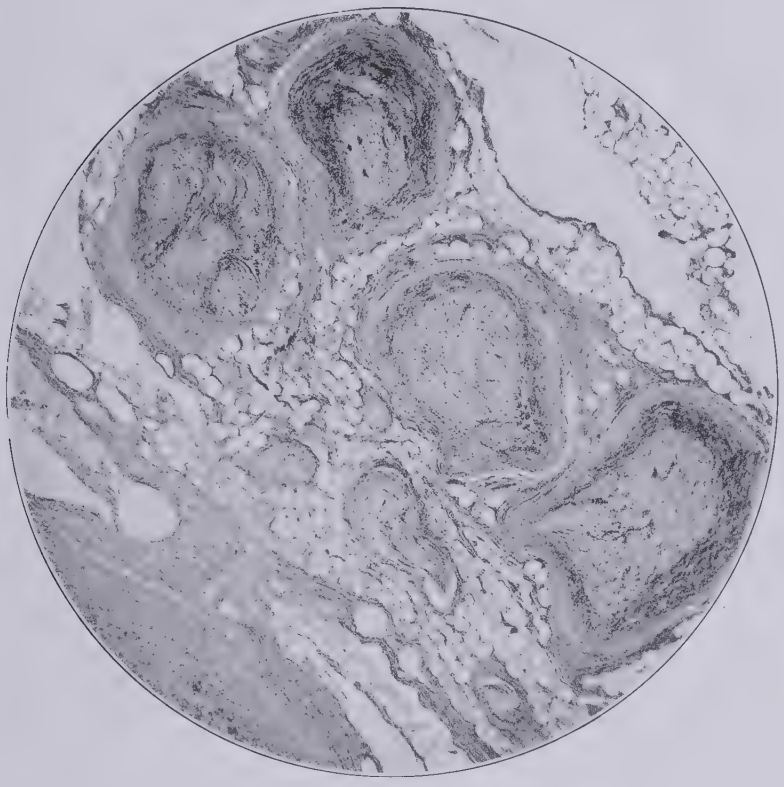


FIG. 34.—Interstitial neuritis.

The myelin becomes turbid, splits up into fragments and droplets. The axis-cylinder also breaks up into fragments or swells up and becomes liquefied. Extravasated leukocytes pick up the products of disintegration and form fat-granule cells. The neurilemma and its nuclei usually remain intact. The nerve during this time shrinks in volume and looks grayish and translucent, or grayish-red. The nerve finally becomes only a fibrous cord. Changes can be seen in the nerve within forty-eight hours, and by this time its irritability, which was first slightly increased,

is lost. In about two weeks the disintegration of the myelin sheath and axis is practically complete. The peripheral end of the cut nerve shows a loss of nearly but not quite all the fibres as far as its termination. In the central end, the degeneration ascends at first only to the first or second node of Ranvier. Very soon, however, a change occurs in the cell from which the fibre springs. This change is called the *reaction at a distance*, or degeneration of Nissl. Thus when the neuraxon is injured the whole neuron suffers, but the peripheral end far the most.

When a section is made between the spinal ganglia and the cord, the fibres all degenerate toward the cord, and even within it, but the peripheral fibres do not degenerate. Hence the spinal ganglia are the trophic centres of the sensory nerves (see Fig. 35).

Degeneration occurs in the motor nerves, also, when the cells of the anterior horns are destroyed. Hence these cells are the trophic

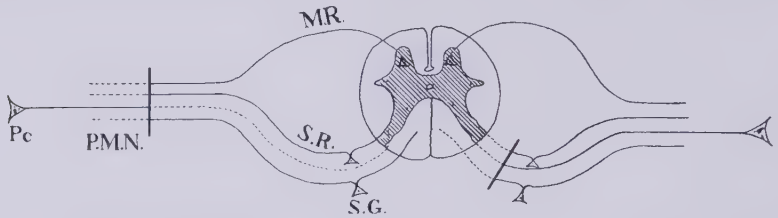


Fig. 35.—Showing effects of section of mixed nerves and sensory root. *PMN.*, Section through mixed nerve; *MR.*, motor root; *S.R.*, sensory root; *S.G.*, spinal ganglion; *Pc*, peripheral ganglion.

centres for all motor nerves. The process of degeneration takes place at about the same time throughout the whole length of the nerve. The motor end-plates in the muscles are affected a little the earliest. About the cut end, little bulbous tumors may develop, which contain numerous nerve fibrils and connective tissue. The general law is that nerves degenerate in the direction in which they carry impulses, but this is not the whole case, as has been just described. If the injury to the nerve is permanent, a slow decay affects the whole neuron. Supposing for example a motor nerve is injured or inflamed at the point *D*. Immediately a degeneration takes place along the parts below to *T*, and in a few days a slight degeneration takes place in the cell-body *C* (see Fig. 36).

Within *the central nervous system* degeneration also occurs mainly in the direction of the nerve impulse. The axis-cylinder is first affected. There is sometimes a preliminary swelling or hypertrophy of this axis-cylinder. Degeneration with calcification of the nerve-fibres sometimes occurs. In associative or commissural fibres the degeneration extends only part of the length of the nerve.

3. Neuritic and toxic nerve degeneration. This form occurs in connection with neuritis, and will be described under that head. Its chief

characteristics are that the degeneration attacks the nerve in segments, that the axis-cylinders are not so much affected, and the myelin breaks up into small fatty droplets instead of into large masses. The same general laws apply to it as to Wallerian degeneration.

Degenerative processes in the non-medullated nervous fibres have been observed in the fine fibres of the cornea and in the submucous and mesenteric plexuses of the alimentary tract.

Regeneration of nerves is a process that usually follows degeneration. It occurs only in peripheral nerves—very little, if at all, in the nerves of the central nervous system of mammals. It is an unique process, in that the nerve is the only specialized tissue that can grow again after being destroyed. Regeneration occurs whenever the trophic centres are healthy, when the mechanical obstacles to a union of the divided fibre

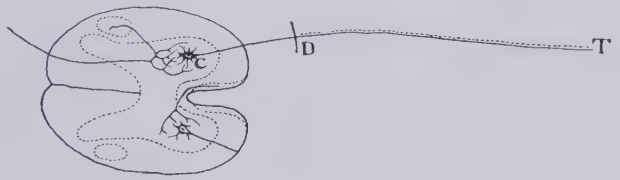


FIG. 36.

are not too great, and when the peripheral nerve is not too completely atrophied. It occurs most quickly, therefore, when the cut ends are carefully apposed and when the separation has not lasted for a long time, *i.e.*, for years. It progresses always from the central end toward the periphery. The fibres of the central stump grow out into the degenerated peripheral fibre. Union by *first intention* or *second intention* never occurs. Human nerves cannot be made to unite physiologically, but only anatomically.

Regeneration may be complete in a few months in short nerves. In the sciatic it may take one or two years. When regeneration takes place, the axis-cylinders of the central stump swell and divide into a number of new cylinders which pierce or creep around the intervening tissue, enter in bundles the peripheral nerve, and become inclosed in new myelin sheaths and neurilemma.

GENERAL SYMPTOMS

The dominant symptoms occurring in the distribution of the peripheral nerves are paralysis and atrophy of muscles in a greater or less degree together with sensory disorders. But since the mixed nerve carries with it some vasomotor and secretory fibres, there may also be congestion, œdema, and dryness or sweating of the skin. When a peripheral motor neuron is

damaged only to a moderate extent or is irritated by any agent, twitchings of the muscle (myokinia) may occur. But direct irritation of the peripheral motor nerve does not cause spasms.

NEURITIS AND MULTIPLE NEURITIS

The common disorder of peripheral nerves is inflammation or neuritis. When this inflammation affects many nerves it is called multiple or polyneuritis. When it affects one nerve or nerve group it is called mononeuritis. The terms plexus neuritis and root neuritis may indicate more specifically the location of the disease.

The following table shows the classification on an etiological basis of the different types of neuritis:

Mono-neuritis
- Traumatic neuritis
- Reflex joint neuritis
- Occupation neuritis
- Rheumatic, infectious and toxic neuritis.

Multiple neuritis
- Infectious neuritis
  - Beri-beri, diphtheria, sepsis
- Toxic neuritis
  - (*a*) Endogenous, diabetic, cachectic, puerperal and senile, etc.
  - (*b*) Exogenous, alcohol, CO, CS2 sulphinal, lead, arsenic, mercury, etc.

MULTIPLE NEURITIS—POLYNEURITIS

Multiple neuritis has been divided into many types in accordance with the cause, the location of the process, and the complications.

1. The common type is a motor-sensory one involving all four extremities and due oftenest to alcohol. Here the paralysis is a dominant feature.

2. Sometimes the sensory and ataxic symptoms are dominant, forming a sensori-ataxic type which is quite rare and is due more often to infections and other poisons than alcohol.

3. A pernicious and hemorrhagic type exists but is also rare. It is due to overwhelming toxæmia.

There is also a pure motor neuronitis, multiple in character, due oftenest to lead; and there is a degenerative neuritis of the sensory neurons, especially noted in diabetes.

It seems best not to have too many kinds of multiple neuritis, and in fact the motor-sensory type is the common and real disease; the others are terminal conditions, complications or sequelæ.

The Sensory-motor Type of Multiple Neuritis (*Alcoholic Paralysis, Arsenical Paralysis, Diphtheritic Paralysis*).—This is the common form

of the disease, making up fully 90 per cent. of all cases seen in the country. While sensory and other symptoms always accompany this type, it is the paralysis and atrophy of muscles which are the leading and chronic conditions. It is a malady coming on acutely, running a subacute or chronic course, rarely fatal, and characterized by weakness or paralysis of all four extremities, associated with atrophy, pain, tenderness and various vasomotor, secretory and trophic disturbances. It is a disease pre-eminently due to poisons and infections, alcohol being far the commonest factor.

It occurs oftener in the female, owing to the fact that women are more anaphylactic to alcohol than men, and perhaps also because of the predisposing influence of tea-drinking.

Multiple neuritis is essentially a disease of early adult life. Almost all cases occur between adolescence and the period of degenerative changes, twenty to forty-five. Young children are very rarely subject to it, but cases have been reported occurring in children at the age of seven, nine, ten and fourteen. A few cases have occurred in persons over sixty, but they are only a little less susceptible than children. It occasionally occurs as an epidemic and is then due to some infection. Beriberi is a particular type of toxic multiple neuritis.

The sporadic forms of polyneuritis from alcohol and various poisons and infections occur without much reference to a seasonal influence. Probably more cases occur in spring and fall, owing to sudden changes in temperature. Epidemic influence like that causing cerebrospinal meningitis may increase the number of cases of multiple neuritis. Practically, in this country, the question of drink settles the question of the distribution of polyneuritis. It is rare in the temperate rural districts and smaller towns, and much rarer in native Americans than in foreigners.

Sexual excesses, exposure to cold and wet, insufficient diet, excessive tea-drinking and the presence of tuberculosis predispose to the disease. The same neuropathic tendency leading persons to excesses in alcohol, tea and to suicidal indulgence in arsenic is of some moment in leading to the development of neuritis.

The list of the special and exciting causes is long and includes nearly all infectious fevers, many chemical and autochthonous poisons. The common infections are diphtheria, puerperal and other septic fevers, and endemic infections of unknown origin. Nearly every infectious fever and possibly malaria may be added to the list.

Of chemical poisons alcohol heads the list, causing over two-thirds of the adult cases. Next come arsenic, lead and phosphorus, mercury, copper and trional. *Wood alcohol* is extremely poisonous, and may promptly cause a multiple neuritis. It has, however, a special tendency

to cause optic neuritis and blindness. Even so small an amount as 3 oz. has caused blindness and 5 oz. blindness and death.

Among the autochthonous poisons, diabetes and the metabolic products resulting from starvation and cachexia lead to multiple neuritis.

Among the foregoing causes, arsenic and diabetes produce more often decided sensory symptoms. One attack does not confer immunity, but it generally compels prudence so that second attacks are rare.

Symptoms.—The disease often begins with prodromata lasting several weeks. The patient suffers from numbness, slight pains, and weakness affecting especially the lower extremities. Sometimes a peculiar condition of mental confusion and weakness precedes the attack. Usually the symptoms come on rather suddenly. The patient suffers from pains and tenderness in the legs and feet, and is obliged to go to bed. There may be a fever for a day or two, the temperature rising to 102° or even 104°F., but this is not the rule. The pains and weakness increase. The muscles and nerves are very tender. The fingers, hands and arms are often similarly but less affected. At the same time the skin becomes reddened or slightly œdematous. The muscles of the legs grow weak, and in a day or two the patient is unable to stand. In a week or two there may be a complete loss of power in the anterior tibial muscles and a lesser degree of paralysis in the extensors of the hand (Fig. 37). Nearly all of the leg and forearm muscles become eventually involved. Atrophy sets in at the same time and very severe pains are present. The motor cranial nerves are in rare cases affected, and paralysis of the facial or of the third, fourth or sixth nerve has been seen. When the disease is fully developed, which is within a fortnight, there is paraplegia with foot-drop, some degree of wrist-drop, muscular atrophy, and slight œdema, especially of the feet. The skin reflexes are often, the knee-jerk and elbow-jerk usually, lost. There is some tactile anæsthesia, often with hyperalgesia. Temperature and pain sense are later lessened and slowed in transmission. The anæsthesia occurs in patches or diffusely. Muscle and articular sense are lost in the sensory or pseudotabetic form, and are usually somewhat involved in the ordinary paralytic form. Pain and sensitiveness continue.

The nerves lose their irritability and the muscles show degeneration reaction, partial or complete, the characteristic being a great variability of reaction over different groups of nerves and at different stages of the disease, and an early loss of faradic and lessening of galvanic irritability. There is sometimes retinal hyperæmia and even optic neuritis. Of the visceral nerves, the vagus seems oftenest to show signs of involvement, with rapid pulse and disturbances of respiration. In rare cases the phrenic nerve is involved. Three such cases were observed in the wards of Bellevue Hospital in two years. The sphincters are rarely affected

and then only for a few days. In such cases there is, perhaps, involvement of the cord or of the abdominal and pelvic splanchnics.

In alcoholic and occasionally in other forms of neuritis, mental symptoms, such as a low, muttering delirium, are very often present, and occasionally a well-marked confusional insanity develops. (Korsakoff's psychosis.)

The disease usually reaches its height in a week or two and then starts on a chronic course; but it sometimes happens that exacerbations occur, or that a paralysis and atrophy progress for several weeks before regression begins. In alcoholic cases there is often great general prostration; the patients lie for several weeks in a delirious condition, and finally develop pneumonia and die.

Fig. 37.—Alcoholic mutiple neuritis with foot-drop and wrist-drop.

In *diphtheritic neuritis* some of the eye and throat muscles (ciliary muscle and soft palate) are involved, while the extremities are usually but slightly or temporarily affected and the sensory symptoms are few.

Some further details should be added.

Motor Symptoms.—The characteristic paralysis of multiple neuritis is a quadruplegia, all four extremities being involved. The special characteristic is the foot-drop, which is indicative of alcoholic neuritis just as wrist-drop is of lead palsy. The paralysis is typically a peripheral one. It affects the feet and legs, hands and forearms. It usually involves the anterior tibial muscles more than the calf muscles, but sometimes the reverse occurs. The muscles become wasted and flabby. They soon lose, in bad cases, all reaction to faradism, and they require a strong galvanic current to produce a contraction. In anterior poliomyelitis, on the other hand, the diminution in galvanic irritability comes on only after weeks or months. Hence an early loss of galvanic as well as faradic reaction is an important sign of neuritis. As the nerve and muscle recuperate, the galvanic irritability increases. After a time, if the paralysis is great, contractures occur. The feet are extended, the wasted

legs are flexed on the thighs and are almost immovable, and the patient's condition is one of pitiable helplessness.

Sensory Symptoms.—Numbness, hyperæsthesia, severe pains (dull and sharp), burning sensations, great tenderness all occur and are very marked symptoms. They are felt mostly in the feet, legs and hands. Hyperæsthesia is usually followed by anæsthesia to touch and somewhat to pain and temperature. The transmission of these latter two sensations is delayed. The anæsthesia sometimes occurs in patches, at other times diffusely over foot, leg and hand. Muscular and articular anæsthesia are common, and in the sensory form are the dominant symptom, causing an ataxia of gait and station. The other special senses are not affected except in rare cases in which there is optic neuritis.

Vasomotor and Trophic Symptoms.—There is often œdema, sometimes redness of the skin; occasionally the epidermis of the soles and palms peels off. Glossy skin and profuse perspiration are rare. Eruptions and ulcers do not occur.

Mental Symptoms.—The most common mental disturbance is that so often seen in acute alcoholism, viz., clouded consciousness and a low muttering delirium. This is associated with great general vital depression. If a true insanity develops it is, as a rule, of the type known as Korsakoff's psychosis. The characteristic symptoms are a curious degree of forgetfulness and disorientation, together with many and varying hallucinations and delusions rapidly succeeding each other. These often relate to the pains and paræsthesia from which the subjects suffer. They think that there are gloves on their hands or that something is on their feet. They often think that they have been out walking or riding. They are talkative, incoherent and sleepless.

Organic Centres.—The bladder is occasionally affected for a short time, the other centres not at all. This freedom from involvement of the sphincters is an important characteristic of the disease in distinguishing it from myelitis.

The *blood* shows a moderate degree of leukocytosis in uncomplicated cases.

From the foregoing it will be seen that the dominant symptoms are paræsthesia, pains (burning, lancinating and dull), muscular tenderness, some anæsthesia, paralysis affecting especially the lower extremities and causing *foot-drop*, muscular wasting, with degeneration reactions; with no involvement of the sphincters; sometimes peculiar mental disturbances.

The sensori-ataxic or pseudo-tabetic type of multiple neuritis is caused less often by alcohol and more often relatively by diabetes and the metallic and infectious poisons. Arsenic given medicinally in doses of one-sixth of a grain or more may cause such a neuritis. Multiple neuritis from lead is not often seen in painters who suffer only mainly from wrist-

drop, but usually occurs when the poison is taken in large doses. The general course of sensory neuritis is much like that of the paralytic form, but there is less paralysis and, on the other hand, there are more of the burning, tearing pains, a greater degree of anæsthesia, with a very decided muscular anæsthesia causing symptoms of a subacute locomotor ataxia. The paresis, muscular wasting, trophic changes, such as shedding of the epidermis and the electrical reactions, serve to distinguish the disease. A facial paralysis sometimes complicates this type.

Endemic and Epidemic Types (*Beri-beri or Kakke, Ignipedites, Acrodynia, Malarial Multiple Neuritis*).—Beri-beri or endemic multiple neuritis is seen in this country rarely, and only by accident. Beri-beri is the Indian name; kakke, meaning "the leg disease," is its Japanese name. Ignipedites is a name given by Indian physicians to probably the same disease. French physicians gave the name of "acrodynia" to an epidemic disease which prevailed in France and the Crimea in the early part of the last century. It was probably multiple neuritis. Epidemic multiple neuritis has been observed in two State hospitals in this country, in Alabama and Arkansas. It has also been observed in epidemics in New York, Connecticut and Vermont. The disease is due mainly to a monotonous and more or less exclusive diet of unpolished rice. It may be produced also by a monotonous diet of other starchy substances, such as wheat; over-crowding, bad hygienic conditions, heat, moisture and exposure favor its development.

There are various types of this disease, in some of which the neuritic symptoms seem subordinate to those of other organs. The forms described are:

The paraplegic or dry, the dropsical or wet, and the mixed and very acute forms.

The symptoms generally resemble those of multiple neuritis, as already described, plus œdema, extensive serous effusions, serious heart weakness and gastro-intestinal disorders. I have observed a case of undoubted endemic neuritis (beri-beri) which ran precisely like an ordinary multiple neuritis. The paralysis affects especially the lower extremities, but in beri-beri there seems to be an especial tendency also to involvement of vasomotor and visceral nerves. The disease runs a course like other types of toxic neuritis. It can be cured by feeding with rice bran, adding other substantial food.

Malarial Multiple Neuritis.—It is likely that "malarial" multiple neuritis is only a form of beri-beri which is not rare in some of the islands of the West Indies.

Acute Pernicious Multiple Neuritis—*The Heine-Medin Disease.*—There is a form of multiple neuritis which comes on suddenly, progresses rapidly, and causes death in a few days or weeks. These cases usually

show the ordinary symptoms of neuritic paralysis, with final involvement of the cardiac and respiratory nerves, causing death. The neuritis is interstitial and hemorrhagic. The disease is due to an infectious poison which overwhelms the system before it has time to set up any inflammation or organic change. In these cases the anterior-horn cells of the spinal cord are also involved, and the disease is one that attacks the whole peripheral motor neuron. It is probable that Landry's paralysis and this severe form of multiple neuritis are due to the same cause, and that they are allied to, if not one phase of, acute anterior poliomyelitis forming a part of the Heine-Medin disease. This is the name given to the infection which causes the above-mentioned diseases as was as encephalitis.

Pathology.—In multiple neuritis the disease affects the periphery of the nerves most, and extends up, very rarely reaching the roots. The

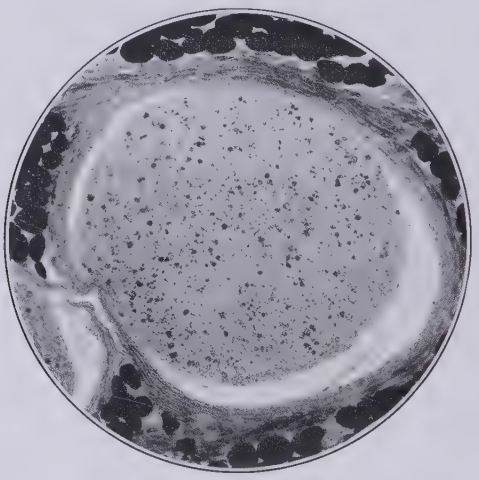


Fig. 38.—Alcoholic neuritis parenchymous degeneration.

anterior tibial and musculo-spiral nerves on the two sides are oftenest and most diseased. The process when mild in grade resembles a secondary degeneration following section of the nerve (Fig. 38). In severer cases there is evidence of interstitial inflammation as well as degeneration. This process, however, varies in degree at different points of the nerve's course. Hence it has been called segmental or disseminated neuritis. In some of these cases and in all acute pernicious cases there is still more interstitial inflammatory change; small hemorrhages occur, exudation takes place, and collections of leukocytes about the vessel walls and among the nerve-fibres are seen (Fig. 39). The muscles supplied by the diseased nerves undergo atrophy. This is usually simple and non-inflammatory. But sometimes there is an interstitial myositis with exudation

compressing the fibres (Senator). If the disease progresses, the nerve-fibres degenerate and their place is taken by connective tissue, and the same process occurs in the muscles.

The spinal cord when examined by the help of Nissl and Marchi stains shows some involvement. The anterior-horn cells undergo the same degeneration as that which occurs when the nerve is cut across, and slight areas of degeneration are found in the posterior and lateral

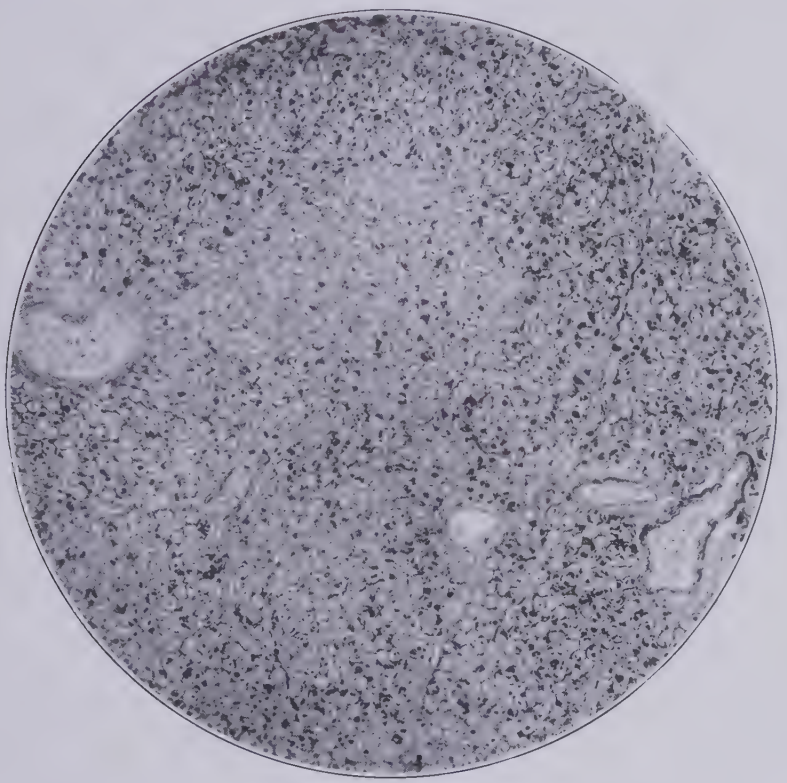


Fig. 39.—Arsenical neuritis (parenchymatous) spinal nerve root.

columns. The changes are very slight compared with those in the nerves, and in the writer's experience are secondary.

It will be seen, therefore, that in multiple neuritis there may be: (1) simple degeneration; (2) parenchymatous inflammation with some evidences of interstitial neuritis (degenerative neuritis); (3) decided interstitial neuritis with degeneration of nerve-fibres. The differences depend on the intensity of the poison.

Diagnosis.—Multiple neuritis must be diagnosticated from diffuse or transverse myelitis, anterior poliomyelitis, locomotor ataxia, spinal

meningitis, and hemorrhage, and from Landry's paralysis. Practically, diffuse myelitis is the disorder from which it has oftenest to be distinguished. From this it is recognized, first, by investigating the cause and onset. Neuritis begins more slowly and with sensory prodromata; it affects the legs and feet, especially the extensors, and if it ascends it skips the hips and trunk and attacks the forearms. There is more muscular atrophy than in myelitis; the knee-jerks are absent. It progresses more slowly, and after four or eight weeks gradually regresses.

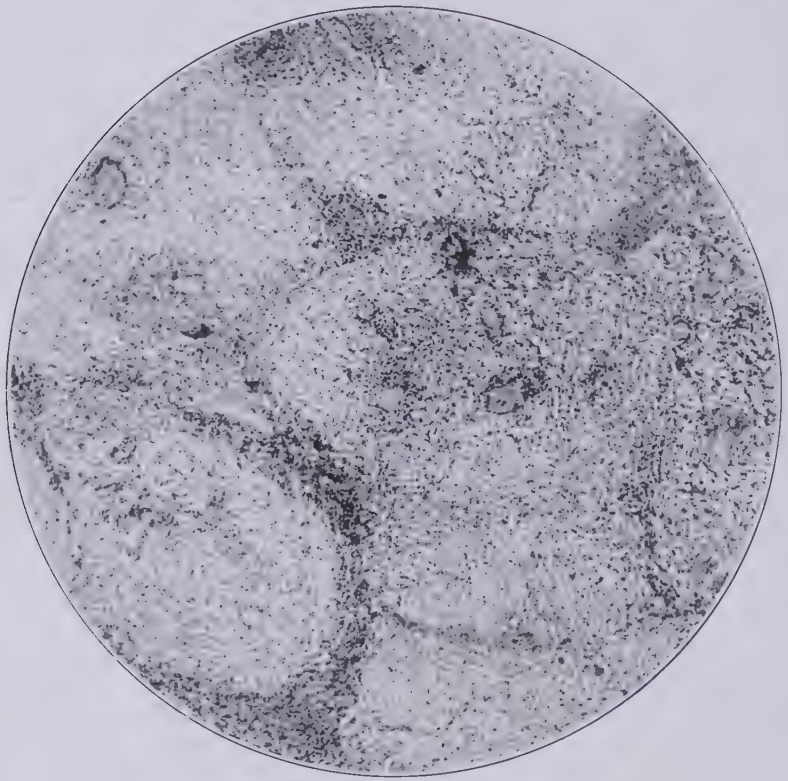


Fig. 40.—Interstitial neuritis, cross-section of 3d cranial nerve.

Electrical degeneration reactions are more varied and decided. There is tenderness over the muscles and nerves and peculiar burning, darting pains. The cutaneous anæsthesia, if present, is not so extensive and complete, as a rule, while muscular anæsthesia is more decidedly marked. There is very rarely involvement of the sphincters nor are there bedsores. There may be belt-like constrictions felt round the extremities, but not around the waist. The gradual improvement of the paralysis and atrophy and eventual recovery confirm the diagnosis of neuritis.

The presence of neuritis of the cranial nerves would strengthen the theory of a general neuritis.

From poliomyelitis it is distinguished by the presence of pain and other sensory symptoms, the early fall in galvanic irritability, the age of the patient and the etiology.

From locomotor ataxia neuritis is distinguished by its more rapid onset, the presence of paralysis and atrophy of muscles, paresis, with degeneration reactions, and the absence of involvement of the organic centres and pupils.

Spinal hemorrhage usually leads soon to a secondary diffuse myelitis easily distinguishable from neuritis by the characters above given. Here there is also usually pain in the back. Spinal meningitis is associated with characteristic pain, tenderness and stiffness along the back. Acute ascending paralysis in its typical form shows but very slight sensory disorders, and no wasting or change in electrical irritability.

The complication of multiple neuritis and myelitis or posterior sclerosis is possible, but is very rare. In the former case the ordinary symptoms of myelitis are added to those of neuritis. In locomotor ataxia there is often some nerve degeneration and occasionally neuritis. The nerve degeneration probably causes only slow atrophic changes and paresis; the neuritis causes pains, anæsthesia, skin eruptions and local trophic disorders.

Prognosis.—Alcoholic multiple neuritis is a serious disease, because of its associated conditions. Nearly one-half of my hospital patients have died, mainly because they continued the use of alcohol after paralysis appeared. They do not die of neuritis, but of alcoholism, pneumonia or of phthisis. Other forms of neuritis rarely cause death. The great majority recover almost completely. It may be from six months to two years before all symptoms disappear. The average time is about a year.

Treatment.—The patient needs, first of all, rest in bed. The limbs are often extremely tender and the patient's pains excruciating. To relieve these the legs may be painted with menthol and enveloped in cotton batting. In other cases flannels wrung out in hot water and renewed every two hours give relief. Internally phenacetin, antipyrin or other coal-tar products may be given for the pains. Fluid extract of ergot in doses of ℥i. to ℥ii. repeated in three hours sometimes relieves pain. In the early stages, salicylate of soda in doses of gr. xx. every two or three hours is recommended. If there is a great deal of depression from alcoholic poisoning, strychnine, gr. $\frac{1}{60}$ q. 3 h., and aromatic spirits of ammonia, ℥ss. q. 3 h., should be used.

There is no drug which really cuts short the process. The best measures for this purpose are rest, thorough cleansing of the alimentary tract, abstinence from alcohol and a nourishing diet.

After the acute stage is passed the labile galvanic current occasionally interrupted may be applied, 5 to 10 ma. for ten minutes three times daily. Later, by the sixth week, the faradic current, massage and careful exercise can be given. At this time or earlier (third week), strychnine, iodide of potassium, arsenic in small doses and tonics may be given. In old cases in which a great deal of paralysis and contracture have occurred, forcible extension of the limbs, the use of splints and rubber muscles are needed. With patience and perseverance the worst chronic cases can eventually be brought to a complete recovery.

Complicating Forms of Neuritis and Neuritic Degeneration.—Neuritis and neuritic degeneration complicate many diseases, but they especially mark and modify subacute and chronic rheumatism, locomotor ataxia, diabetes, paralysis agitans, wasting diseases and old age.

A *neuritic* degeneration almost always affects the nerves in the neighborhood of an old rheumatic joint. The chief result of this is to produce wasting and some paresis of the muscles moving the joint (Pitres and Vaillard). The process is a reflex atrophy (see Arthritic Muscular Atrophy).

In locomotor ataxia parenchymatous nerve degeneration is very often present. It does not produce the cardinal symptoms of this disease. It does, however, cause some of the anæsthesia, paræsthesia, muscular atrophy, skin dystrophies and visceral crises.

In diabetes the neuritis takes the form of the sensory type of multiple neuritis, and causes paræsthesias and neuralgias somewhat like those of locomotor ataxia. The patient has sciatic, intercostal and other neuralgic pains, burning or numb feet, and sometimes loss of tendon reflex. The upper extremities are rarely affected.

In Wasting Diseases and Old Age.—In various wasting diseases, such as phthisis, cancerous cachexia, long-continued fevers, marasmus, and in senility, a simple parenchymatous degeneration of nerves, with atrophy, occurs (Arthaud, Köster, Jappa). The symptoms caused by these changes are very slight. They contribute to the weakness and wasting. In old age, the atrophy of the nerves is one cause of the lessened sensibility and activity of the skin and its underlying muscles.

TUMORS OF NERVE

Classification and Pathology—Nerve tumors occur as:

1. Nerve hyperplasia.
2. True neuromata.
3. Fibro-neuromata.
4. False neuromata.

1. *Hyperplasia* or hypertrophy of nerve trunks is very rare. Gen-

erally the increase in size is due to increase of the interstitial connective tissue. Sometimes, however, there is an increase in the number of fibres and thickening of the myelin sheath.

2. *True neuromata* are also very rare, and occur almost exclusively on spinal nerves. In some there is an increase in medullary fibres; in others only an increase of non-medullated fibres, *i.e.*, only the axis-cylinders and neurilemma increase. They occur either singly or multiply.

True neuromata are usually small, ranging from 1 cm. (two-fifths of an inch) to 6 cm. in diameter. They may be much smaller or larger. They are usually few in number, but may be very numerous. Gowers estimates in one case that as many as 1000 were present. Even larger numbers have been observed. They rarely cause serious symptoms, but may produce local pains and paræsthesias.

3. *Multiple fibro-neuromata,* when numerous, constitute a condition known as *neurofibromatosis* (Recklinghausen's disease).

This is a rare condition, but one of importance because sometimes these tumors develop on cranial nerves, gradually increase in size and become practically brain tumors. The following description is from an article upon this subject by Fraenkel and Hunt.

The tumors occur in varying size from that of a pin's head to that of a goose egg. In some cases the manifestation of the disease is expressed by a solitary growth upon one nerve, and in other cases several hundred growths have been present. They occur on all cerebrospinal and sympathetic nerves except on the optic and the olfactory. The acoustic nerve has been shown to be particularly liable to the formation of fibromata and fibrosarcomata. The origin of these tumors is usually from the endoneurium, rarely from the perineurium and exceptionally from the epineurium. They are situated either centrally within the nerve or eccentrically on the periphery of the nerve. The general conformation of the tumors is spherical or elliptical and in generalized cases the nodular outgrowths give the nerve a convoluted appearance, sometimes resembling a rosary.

Histologically, the fibroma nervorum is made up of connective-tissue elements, which present all the variations in structure between the fibroma molle and the fibroma durum.

The neurofibromata are usually slowly growing tumors of a benign nature. Sometimes, however, they undergo a retrograde metamorphosis and show cystic, fatty or myxomatous degeneration or they undergo sarcomatous transformation, assuming a malignant character.

Neurofibromatosis depends often on a hereditary or congenital disposition. It is frequently associated with more or less mental impairment, imbecility, and somatic stigmata of degeneracy. Scrofulosis and

tuberculosis are often found in the histories of the cases. The tumors are not painful and cause no symptoms when confined to the skin.

FIG. 41.—Neurofibromatosis. (*Fraenkel and Hunt.*)

Neurofibromatosis may show itself in one of the following ways: (1) Tubercula dolorosa (painful tubercles, Wood); (2) multiple neuro-

fibromata of the skin (molluscum fibrosum, Recklinghausen's disease); (3) neuromata or neurofibromata of a single peripheral nerve; (4) neuroma plexiforme, pachydermatocele, and elephantiasis neuromatodes; (5) generalized neurofibromatosis; neurofibromata of the skin, cerebrospinal and sympathetic nerves.

3. Neurofibromata of individual peripheral spinal nerves (isolated neuromata) are surgically well known. They express themselves clinically by paræsthesias and progressive motor weakness of the peripheral nerve type, and are of good prognosis when recognized. They occur most frequently on the median and sciatic nerves; they are rarely without symptoms and are prone to give rise to considerable hyperæsthesias and little paralysis.

4. *False Neuromata.*—This term is applied to the various nerve tumors in which a fibroma, myxoma, glioma, sarcoma, carcinoma or syphiloma grows upon or in the nerve. The fibroneuroma as above described is really a false neuroma and is the common form; glioneuroma has been observed on the auditory nerve. Syphiloma occurs only on the intracranial or intraspinal nerves. Carcinoma of nerves may be primary, but is generally secondary, and is of scirrhous or medullary type, rarely the colloid. Leprous neuritis sometimes forms neurofibromatous swellings. A few cases have been observed of multiple malignant neuromata. Trauma and hereditary influence are the etiological factors. The great nerve trunks are oftenest affected, the median, sciatic and crural ranking first. The tumors start from the perineurium; they are at first spindle-shaped, and may grow very large. Sarcomatous cells are oftenest found in them; but they may be myxomatous, fibromatous or mixed.

Etiology.—Three general causes exist for the production of neuromata, viz.: 1. A hereditary or a neuropathic predisposition, which tends to cause the true, the multiple, and the plexiform neuromata. 2. Injuries, surgical operations; these cause especially the fibroneuromata of which the *amputation neuroma* is an example. 3. Diathetic, *e.g.*, tuberculous influences, and whatever produces the various tumor formations, sarcoma, carcinoma, form the third etiological factor. Neuromata of the plexiform type are often congenital. Multiple neuromata may develop early in life. Men are far more subject to multiple neuromata than women.

Symptoms.—Neuromata often cause no symptoms. Perhaps the most frequent evidence of their presence, however, is pain and some tenderness. The pain is exacerbating, and may be stopped sometimes by pressure on the nerve above the tumor. Paræsthesia, anæsthesia, paralysis, and reflex spasm may be present. Some forms of intractable headache are possibly due to multiple neuromata. A neuroma upon one of the cranial nerves, usually the acoustic, may develop into and cause

the symptoms of a brain tumor. Hence the presence of neurofibromata of the skin with brain symptoms should always suggest the possible presence of an intracranial growth. Multiple and plexiform neuromata cause symptoms less often than a single larger neuroma. A neuroma on the pneumogastric or other splanchnic nerve may cause severe symptoms.

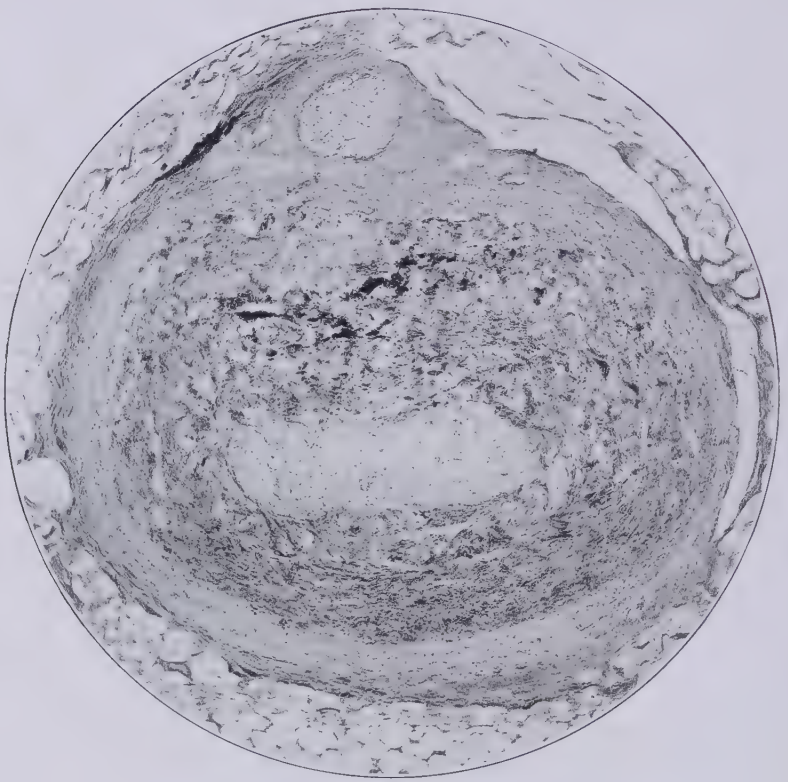


Fig. 42.—Neuro-fibroma, Recklinghausen's disease.

Multiple (true) neuromata may last for years and cause no serious inconvenience. Malignant neuromata cause always such symptoms as would naturally follow irritation and compression of a nerve.

The *treatment* of neurofibromata and of nerve tumors generally is usually purely surgical when any treatment is indicated.

CHAPTER VIII

MOTOR DISORDERS OF CRANIAL NERVES

The general distribution of the paralyses from injury or disease of the different motor nerves is shown in the accompanying table of cases. I am indebted to my friends Dr. Wm. P. Wilkin and Dr. Alexander S. Leverty, for the larger part of the work of compilation:

Cranial-nerve paralyses (including 19 occurring in locomotor ataxia. .	206
Spinal-nerve paralyses.	377
Multiple neuritis	114
	697

The cranial nerves having motor function are paralyzed in about the following frequency:

Facial, 136; Oculo-motor, 52; V, X, XI, 3.

Taking all motor nerves, cranial and spinal, one finds that the brachial plexus and its branches are oftenest affected by paralysis, next the seventh cranial nerve, then the ocular nerves, and last the lumbar and sacral plexus. This represents the frequency in a neurologist's experience. In general practice and especially in surgical practice there would be fewer cases of cranial-nerve disease and more of lumbar and sacral palsies.

THE OCULAR MUSCLES

Anatomy.—The motor nerves of the eye are:

(*a*) The third or oculo-motorius, supplying the internal, superior and inferior recti, inferior obliquus, the levator palpebræ, the ciliary muscle, and constrictor of the iris.

(*b*) The fourth or trochlearis supplying the superior oblique.

(*c*) The sixth or abducens, supplying the external rectus.

(*d*) The sympathetic, consisting of fibres from the cervical cord to the dilators of the iris, to its blood-vessels, and to the unstriped portion of the levator palpebræ and to the unstriped musculus orbicularis.

The third and fourth nerves arise from a series of nuclei in the floor of the aqueduct of Sylvius. They leave the brain at the anterior edge of the pons. They run in the cavernous sinus and enter the orbit through the sphenoidal fissure.

The sixth nerve arises from a nucleus in the floor of the fourth ventricle. It emerges at the posterior edge of the pons, runs in the cavernous sinus, and enters the orbit through the anterior lacerated foramen.

The nuclear gray matter from which these nerves arise is made up of a series of nests of cells and each pair supplies a different set of muscles of the eye, as shown in the diagrams (Figs. 44 and 47).

The nucleus of the sixth lies farther back in the floor of the medulla, but it belongs to the same serial deposit of gray matter and represents the continuation of the anterior horn of the spinal cord (Fig. 48).

The motor nerves of the eye, third, fourth and sixth, are closely connected with each other and other nerves by a long commissure, the *posterior longitudinal bundle.*

The fibres of the third and sixth nerves pass to their nuclei on the same side, then decussate and pass up in the inner part of the crusta to the frontal part of the præcentral convolutions of the cortex. A few fibres decussate and enter the nuclei of the opposite side. They are connected with the internal rectus nucleus.

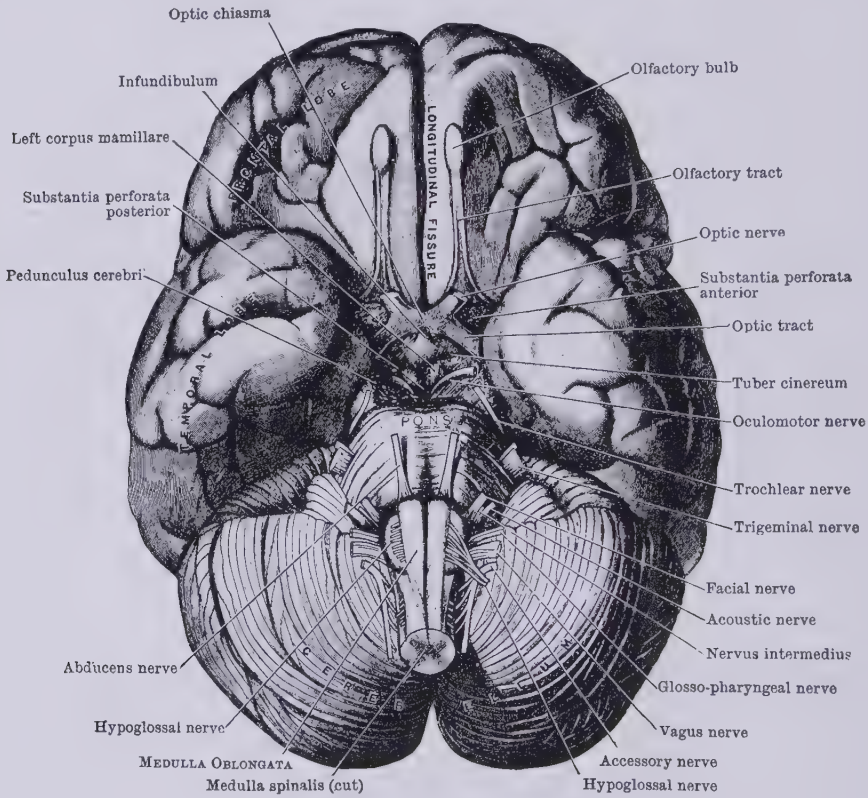


Fig. 43.—Showing the origin of the nerves at the base of the brain.

The fibres of the fourth nerve almost entirely decussate, running forward a long distance before they finally reach their nuclei. Thus it appears that the fourth is the only cranial nerve except the optic which largely decussates before reaching its nucleus. However, those fibres of the third which supply the internal rectus also decussate, as already stated.

The arrangement and composition of the nuclei of the oculo-motor nerves as given by Bing is as follows:

1. A small-celled lateral nucleus, the nucleus of Edinger and Westphal.
2. A large-celled lateral nucleus.
3. A small-celled mesial nucleus.

Of these the longest is the large-celled lateral nucleus. It contains, ranged one behind the other

(*a*) The centre for the levator palpebræ superioris.
(*b*) The centre for the rectus superior.
(*c*) The centre for the rectus internus.
(*d*) The centre for the obliquus inferior.
(*e*) The centre for the rectus inferior. (See Fig. 44.)

The fibres from (*a*) and (*b*) arise exclusively from the nucleus of the same side, those from (*e*) exclusively from that of the opposite side, while those from (*c*) and (*d*) arise from the nuclei of both sides.

The mesial nucleus is the centre for accommodation. The Westphal-Edinger nucleus innervates the *sphincter pupillæ*.

The mesial and lateral small-cell nuclei send fibres which pass first to a ganglion before going to the muscles they supply and they belong to the mid-brain autonomic system.

In order to understand the peculiarities of eye palsies, to be described later, the relations of the sixth to that nucleus of the third nerve which innervates the internal rectus must be understood. In turning the eyes to one side, these two nuclei and their nerves act together, causing the external rectus of one eye and the internal rectus of the other to contract at the same time. The impulse from the brain which does this decussates and acts first upon the sixth, and through this upon the external-rectus nucleus of the same side. The impulse from this nucleus then goes to the third-nerve fibres of the same side and thence to the internal rectus. This can be better understood by the diagram, Fig. 45.

Thus lesions in the brain at (*a*) cause paralysis of the sixth nerve of the opposite side and internal rectus of the same side. The eyes turn toward the side of the lesion.

Lesions in the pons at (*b*) cause paralysis of the sixth on the same side and internal-rectus nucleus of the opposite side. The eyes turn away from the side of the lesion.

The movements of the eyeball are made by the simultaneous action of several muscles. Most of them act as their names indicate. But the oblique muscles help to depress or elevate, and then help to rotate in or out according as the internal or external rectus is acting.

The cortical centres for the eye muscles are not positively known. Lesions in the inferior parietal lobule sometimes cause disorder of the third nerve, but the dominant centres are in the prefrontal lobe at the base of the upper and middle frontal convolutions.

As the optic nerve is the special sensory nerve of the eye, so the third, fourth, sixth and part of the seventh nerves are the motor nerves. By means of the optic nerve and its receptive and refractive apparatus, the form, color, movement and, to some extent, relations and distance of objects are determined. The motor nerves adjust the eye to near and

distant objects, inform us as to size and distance, and enable us to follow moving objects and to shift the gaze readily. They also assist in protecting the eye against injury.

General Symptoms.—It is impossible always to disassociate diseases of the oculo-motor nerves from those involving their nuclei. Hence we must study here really the affections of the whole neurons. These are:

(1) Paralyses or ophthalmoplegias, which may be acute, chronic, or progressive; (2) pareses or myasthenic states, called ordinarily muscu-

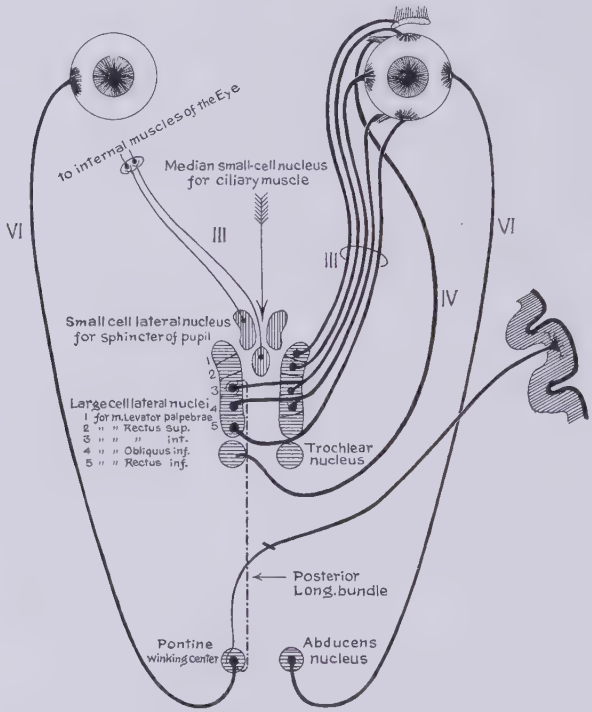


FIG. 44.—The nuclei of the motor nerves of the eye. (*Bing.*)

lar asthenopias; (3) spasms, such as strabismus, nystagmus, and blepharospasm; (4) reflex disorders.

There are many special terms which are used to indicate the peculiar effects of various paralyses and spasms of the ocular muscles and nerves, and some of these I will define here:

Erroneous protection is a condition in which the patient is unable to judge exactly of the relation of external objects to the body; for this relation is determined by the movements of the ocular muscles, and, these being weak, wrong sensations are conveyed to the brain. Vertigo may result from this disturbance of muscular sensation.

Diplopia or *double vision* is a condition due to the erroneous sensation resulting from eye-muscle palsy, and to the fact that the images of the object fall upon non-corresponding retinal fields. Diplopia is simple or homonymous when the false image is seen on the same side as the affected eye. When a red glass is placed over this eye two images are seen, the red one being on the side of the eye involved. Diplopia is heteronymous or crossed when the false image is on the side opposite to the sound eye.

Conjugate deviation of the eyes is a condition in which both eyes turn strongly to one or the other side. It may be paralytic or spasmodic. The mechanism is a complicated one and not perfectly understood. In general, destructive lesions of the brain cause a paralytic

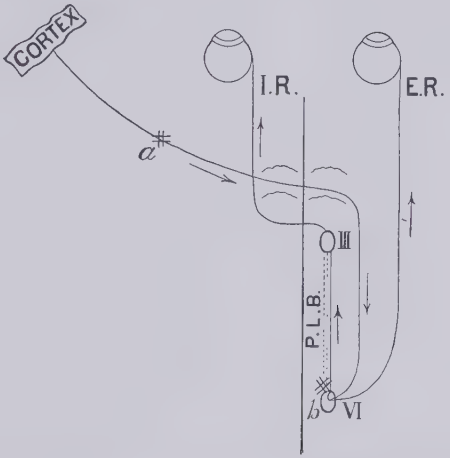


FIG. 45.—Diagram showing the probable relations of the nuclei of the sixth and of the internal rectus branch of the third to the brain. *P. L. B.*, Posterior longitudinal bundle.

deviation toward the side of the lesion, and irritative or compressing lesions the opposite effect. Destructive lesions in the pons cause a deviation away from the side of the lesions. The palsy then involves the sixth cranial nerve and the branch to the internal rectus from the third. The sixth-nerve nucleus is the dominant one, and impulses from the brain go to it first (see Fig. 45).

In diseases of the motor nerves of the eye it is found that the paralyses occur in various ways, which may be best grouped as follows:

Ophthalmoplegias

1. Paralyses of the third nerve. } Acute.
2. Paralyses of the fourth and sixth nerves. } Chronic.
3. Progressive paralysis of all or part of these nerves.

THE OPHTHALMOPLEGIAS

I. Paralysis of the Oculo-motorius or Third Nerve—Etiology.—The commonest causes are "exposure to cold" (which means some simple infection), and syphilis. Other causes are basal meningitis, intracranial tumors, injuries, compression from orbital tumors, the diphtheritic poison; excessive use of tobacco and alcohol, of morphine, or other drugs may be predisposing causes. Temporary palsy sometimes occurs in migraine or it may take the place of an attack of migraine. Partial or temporary palsies also occur in locomotor ataxia, in diphtheria, in certain primary muscular atrophies and in myasthenia gravis.

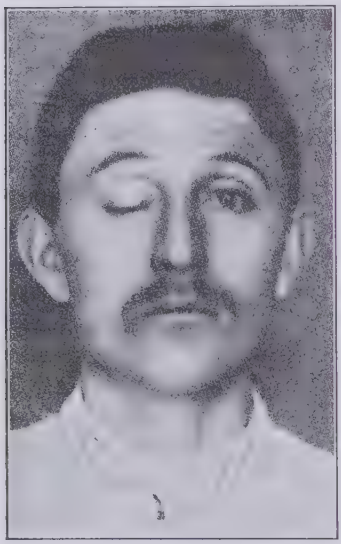


Fig. 46.—Paralysis of 3d nerve with ptosis. (*Wildbrandt and Sanger.*)

There occurs, in rare cases, an acute inflammatory degeneration of the nuclei of the ocular-muscle nerves similar to acute anterior poliomyelitis. This condition has been called "polioencephalitis superior" or upper bulbar palsy.

Symptoms.—When all the muscles supplied by the third nerve are paralyzed, there is dropping of the lid (ptosis, Fig. 46); the eye can be moved only outward, and downward and inward; there is, therefore, divergent strabismus and double vision (diplopia). The pupil is somewhat dilated and does not contract to light, owing to paralysis of the constrictors of the iris; and there is loss of power of accommodation, so that the patient cannot read print close to him.

The patient suffers much annoyance from the lid-drop and the double vision, and there are sometimes vertigo and photophobia. Only one nerve is involved at a time, as a rule. The various eye muscles supplied by the third are rarely all attacked. The levator may escape almost entirely; the ciliary muscle and iris may also be but slightly involved; while these latter muscles are never involved alone in ordinary types of the disease. They may be paralyzed alone in diphtheria or in an attack of migraine.

The affection usually runs a subacute course, lasting but a few weeks. Functional palsies last but a few days; syphilitic palsies are usually temporary (one to three weeks), but may relapse or become extremely obstinate. Periodical palsies occur every year or six months or even oftener; they last a few days or weeks and are accompanied at

first by some pain. They continue to recur for years. They may be associated with attacks of migraine and are migrainous equivalents.

In diphtheritic eye palsies the first three or four nuclei of the series making up the origin of the third nerve are oftenest affected, causing paralysis of accommodation, paralysis of the iris and of the internal rectus, the three muscles concerned in accommodating the eye to near objects.

Pathology.—In the rheumatic palsies there is a low grade of peripheral neuritis, and the same is true of most diphtheritic and other palsies of infectious origin. In syphilitic and tabetic palsies there is usually a specific basilar meningitis involving the nerve-roots. The meningitis may be slight or may amount to gummatous deposit. In functional and some periodical palsies there is a vasomotor disturbance causing congestion or anæmia or perhaps simply inhibition of the nuclear centres. Some periodical palsies have been found to be due to small tumors involving the nerve-root. In rare cases there is primary muscular atrophy of the eyeball nerves or primary degeneration of the nuclear centres. The nuclear inflammation forming "polioencephalitis superior" is a disease probably infectious and quite similar to anterior poliomyelitis.

Special Symptoms.—One must determine how extensively and which of the muscles supplied by the third nerve are involved.

If only the levator palpebræ, there is simply falling of the lid.

If the external eyeball muscles are involved, we get:

Limitation of movement of the globe.

Strabismus and secondary deviation.

Erroneous projection.

Double vision or diplopia, which is either simple or crossed.

If the motor fibres to the iris are paralyzed there is immobility and dilatation of the pupil and there is a loss of light reflex. If the light reflex is gone but accommodation persists, we have the so-called Argyll-Robertson pupil.

Iridoplegia without cycloplegia is most significantly present in syphilitic conditions. Iridoplegia may also occur in infections like diphtheria and from toxic states and local disease.

If the motor fibres to the ciliary muscle are paralyzed we have *cycloplegia*, or paralysis of accommodation. It also may occur in syphilis, tabes, paresis and in diphtheria. It sometimes occurs as a symptom in migraine.

Paralysis of Convergence.—The internal recti muscles may be quite normal except that they cannot converge the eye. When this is the case there is also loss of the associated pupil contraction. This form of paralysis sometimes occurs. It is inferred that there are two separate centres

for contracting the pupil. One associated with the light reflex and one with convergent movement (Turner).

Paralysis of the levator palpebræ, causing *ptosis*, is sometimes seen alone. A functional palsy of the lids sometimes occurs in anæmic and nervous people at the time of waking. It is a temporary *morning* or *waking ptosis*. Ptosis is a frequent and early symptom of myasthenia gravis.

Paralysis of the sympathetic fibres of the eye causes the *Horner syndrome*, viz.: contraction of the pupil (myosis) from the unopposed action of the third nerve. There is also a slight retraction of the eyeball, a narrowing of the palpebral fissure, sweating of the face on the affected side and lessened intraocular tension. The pupil does not dilate when the skin of the cheek or neck is irritated, a condition known as loss of oculo-skin reflex and it does not dilate in the dark or respond to an instillation of cocaine. On the other hand the pupil contracts to near vision and convergence. The spinal centre is in the first dorsal and eighth cervical segment. The fibres pass out through the first and second anterior dorsal roots, scend in the cervical sympathetic, pass to the Gasserian ganglion and thence through the first division of the trigeminus to the pupil.

II. **Paralysis of the Fourth Nerve.**—This is a rare affection and not always easily detected. The causes are much the same as those of palsy of the third nerve. The symptoms are slight convergent strabismus when the eye is moved downward and diplopia on looking down. There is defect in the movements of the eye downward and outward.

III. **Paralysis of the sixth nerve** (abducens) is the most frequent of eye palsies, and occurs especially often in syphilis and in locomotor ataxia. It causes convergent strabismus and double vision.

Progressive Ophthalmoplegia.—Besides the palsies already described, there occur certain forms which have a peculiar origin and course. They begin slowly, as a rule, and steadily progress. In some cases only do they reach a certain stage and then remain chronic. They often affect the third, fourth and sixth nerves together. In accordance with the muscles invaded, these palsies are called *external*, *internal*, *partial* and *total*. Thus if those branches of the third nerve supplying the iris and ciliary muscle are involved alone, it is ophthalmoplegia interna; if the other branches are involved, it is called ophthalmoplegia externa.

Definition.—Progressive ophthalmoplegia is a degenerative disease of the nuclei of the motor nerves of the eye. It is in most cases the same disorder as of bulbar paralysis and progressive muscular atrophy.

Etiology.—It develops between the ages of fifteen and forty, but may occur later. The sexes are equally affected. Lead, diphtheria, traumatism, syphilis, appear sometimes to be the cause. It may compli-

cate locomotor ataxia; more often it forms part of progressive muscular atrophy.

The *symptoms* are often not noticed until the disease is well advanced. The vision is not disordered, and there is only a gradual limitation of mobility of the eyeball. A slight drooping of the lids, causing a sleepy look, or a slight squint, usually divergent, is noticed. Then upon examination it is found that the eyes are immovable and cannot follow the finger, except to a slight extent. The peculiar physiognomy which results is known as the "Hutchinson face." The iris reacts to accommodation and light usually. Double vision may be present. Usually the patient accustoms himself to monocular vision. The disease lasts a long time, and it may become stationary. If complicated with progressive muscular atrophy, however, the course is relatively rapid, death occurring from the latter disease in two or three years.

Pathological Anatomy.—In all progressive cases there is a degenerative atrophy of the nuclear cells. In a few stationary cases the anatomical change is that of neuritis.

The *treatment* is that for the disease which it complicates or the condition which causes it. That is to say, it is the treatment for locomotor ataxia, progressive muscular atrophy, syphilis or lead poisoning. Iodide of potassium, strychnine, arsenic, and phosphorus may be given. Electricity is of no value. General tonic measures and rest to the eyes should be employed.

Muscular Asthenopia and Muscular Insufficiencies.—This is a term employed to indicate a lack of equilibrium of the muscles of the eye, as a result of which the visual axes cannot be kept parallel without an effort. This effort is often unconscious, and shows itself only by a ready tiring of the eye on attempting to read or by the production of headaches and cerebral paræsthesias. Examination of the eye by means of prisms reveals the special character of the trouble. The term is usually applied to functional, congenital and neurasthenic weaknesses, not to those due to organic disease or severe toxæmias.

When the eye muscles act normally, the condition is called one of *orthophoria*. When some of the muscles are weak it is called *heterophoria*. There are various forms of heterophoria, viz.: esophoria, a tending of the visual lines inward, from weakness of the externi; exophoria, a tending of visual lines outward; hyperphoria, a tending of the visual line of one eye above its fellow.

The condition is tested in various ways. The simplest is this: Refractive errors having been corrected, a series of prisms is placed over the eye, at first with the base inward, while the patient looks at a candle 20 feet distant. The prisms are increased in strength until the patient can no longer coalesce the images. The degree of prism is noted, and

this indicates the strength of abduction or of the externi. The same process is gone through with for the interni, the base of the prism being out. The externi should overcome a prism of about 8°, the interni one of 23° to 25° or more. There are great individual variations, and there is also considerable variation in individuals.

For details of the technic for testing heterophoria the reader can best consult special works.

Muscular asthenopia is said to cause a disturbance of vision, vertigo migraine, cerebral paræsthesia, and pains in the head, more particularly in the occipital and cervical region. It may be a factor in producing choreic twitchings in the face. In neurasthenic persons it may cause a wider range of nervous symtpoms. It is said to be a factor in causing epilepsy, chorea, and hysteria. The author cannot accept this latter view, and believes that the importance of muscular asthenopia in causing general nervous symptoms is not great. Much of it, if not all, may be relieved after correcting refractive errors by helping the general health of the patient.

The treatment of it, after all myopia, or hypermetropia, or astigmatism, if present, is relieved, consists in building up the general health, in the systematic use of prisms for training the muscles, and the wearing of proper glasses. Sometimes graduated or complete tenotomies are advisable.

SPASMODIC DISEASES OF THE OCULAR MUSCLES

These are: (1) Conjugate deviation from spasm; (2) irregular and associated spasms from convulsive and irritative brain disorder; (3) nystagmus.

Spasmodic conjugate deviation occurs from an irritating lesion of the ocular nuclei or of the brain in its cortical motor and mid-brain tracts. Irregular spasmodic movements occur in meningitis, hydrocephalus, and in lesions involving the semicircular canals. Peculiar associated spasms occur in hysterical attacks. Various spasmodic movements and contractions of individual eye muscles occur from ocular disease, errors of refraction, muscular weakness, and paralysis of certain eye muscles.

Rhythmical spasm or *nystagmus* occurs as the result of hereditary visual weaknesses, of refractive errors of various kinds, in albinoes, and in chronic hydrocephalus. It is found usually in neurotic persons who have ocular defects, in multiple sclerosis, and sometimes in chorea. It occurs also in hereditary ataxia, tumors, especially of the cerebellum, and mid-brain, and in meningitis. It occurs in miners and is called miner's nystagmus.

In nystagmus the oscillation of the eyeballs is usually lateral. It may be brought out when slight in degree by causing the patient to look

steadily to one side. Vertical and a kind of rotating nystagmus sometimes occur, and are due to lesions of the mid-brain and its vestibular connections.

Spasm of the levator palpebræ is sometimes seen and is usually tonic.

The above troubles are all symptomatic, and their treatment depends upon correction of some local disease or serious cerebral lesion.

THE MOTOR BRANCH OF THE FIFTH CRANIAL NERVE

The anatomy of this nerve is described under the head of the neuralgias of the trigeminus.

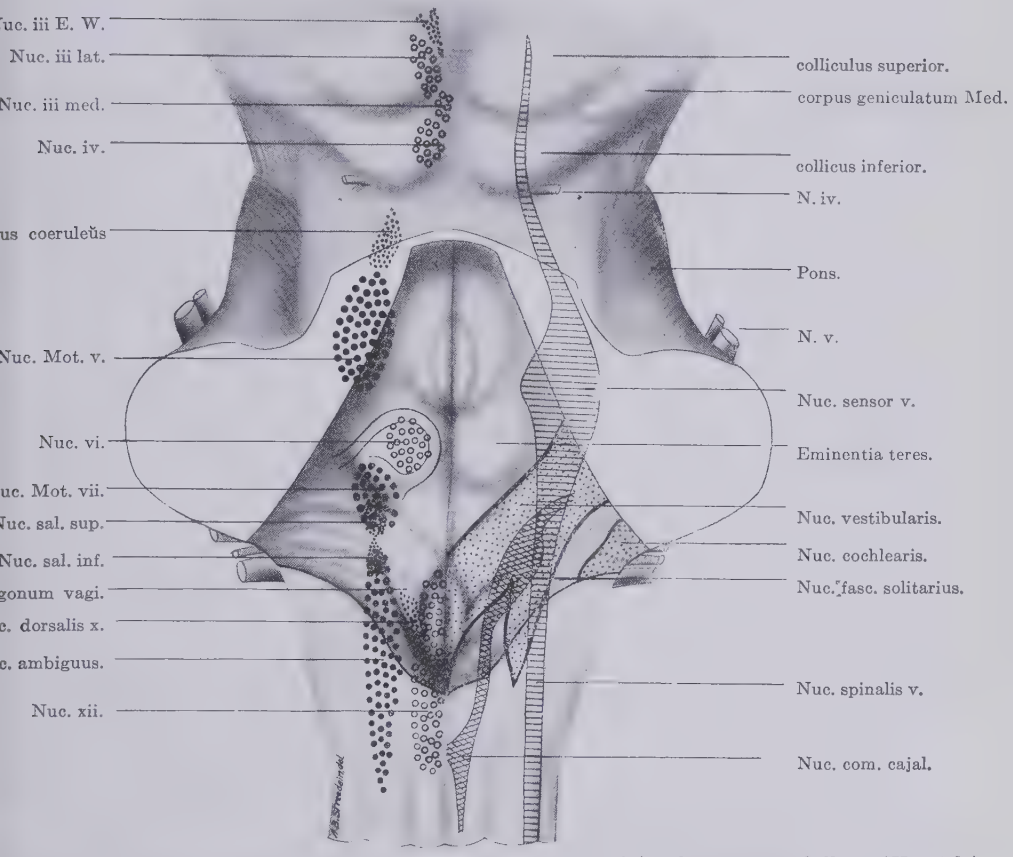


FIG. 47.—Showing the position of the cranial nerve nuclei in the pons-medulla. (*Herrick.*)

The diseases of the motor branch of the trigeminus are rare, and generally symptomatic of some more general disorder.

Trismus (*lockjaw*) is the only important independent affection of this motor nerve. It is a tonic spasm of the muscles of mastication.

Etiology.—It occurs in infants, usually through infection from the umbilicus. It is then known as trismus nascentium. It forms part of the symptoms of tetanus, tetany and rabies. It may be symptomatic of brain disease, and forms one of the manifestations of the epileptic fit and of hysteria. There may be a reflex trismus from irritations of the teeth and jaw and from gastro-intestinal trouble. It may be associated with a trigeminal neuralgia.

Symptoms.—There is, as shown above, a symptomatic, an infectious, a reflex, and a hysterical trismus. In all, the symptoms are very manifest. The jaws are firmly locked and the masseters and temporals stand out. If the disease is unilateral, which is rarely the case, the lower jaw is pushed over toward the sound side.

The *treatment* of both tonic and clonic spasms depends upon the cause. Symptomatically, morphine is to be given, and later the antispasmodics, such as the bromides and chloral. In rheumatic cases, hot applications and diaphoretics are indicated.

THE FACIAL NERVE

Anatomy.—The facial nerve has its primary origin in a single nucleus deeply situated in the lower part of the pons (Fig. 48). It belongs to the same series of nuclei as the vagus, glossopharyngeal, and spinal accessory; in other words, it is a prolongation of the lateral horn of the spinal cord. It receives some fibres from the hypoglossal nerve which supplies the orbicularis oris, in part. The deep facial fibres curve down and out around the nucleus of the sixth nerve (Fig. 48). The cortical origin of the seventh nerve is in the lower part of the præcentral convolution. The upper branch of the facial which supplies the muscles of the upper part of the face has a cortical origin from each hemisphere, so that a lesion of one cerebral hemisphere does not cause its paralysis. The fibres from the cortex pass down through the knee of the internal capsule and enter the crusta at the inner side of the pyramidal tract. They decussate and reach the nucleus.

The nerve has its exit at the posterior edge of the pons, external to the sixth nerve. It has then to take a long course through the internal auditory meatus and Fallopian aqueduct. During its course here it receives communications from the nerve of Wrisberg and from the great superior petrosal nerve. It sends a motor branch to the stapedius muscle in the middle ear and it gives off the corda tympani nerve. The nerve passes out through the stylo-mastoid foramen and supplies all the muscles of the face, the stylo-hyoid, posterior belly of the digastric, the buccinator and the platysma myoid. It does not supply the soft palate or the muscles of mastication. The facial nerve is a mixed nerve and has a sensory part. The fibres of this sensory root arise in the geniculate ganglion. They pass centrally into the medulla to a small nucleus near that of the glossopharyngeal; the peripheral fibres pass in the nerve of Wrisberg and communicate with the motor portion of the seventh.

Motor Function.—As already stated, the facial nerve is a motor nerve for the muscles of the ear and face and for the stapedius muscle, a muscle which antagonizes the tensor tympani.

Sensory Function.—The nerve of Wrisberg and its ganglion contains a few vestigial fibres which supply cutaneous sensation to a small area in the concha and to the posterior wall of the external meatus (J. Ramsay Hunt).

Special Sense.—Fibres of the special sense of taste originating in the medulla pass probably in the second and third branch of the trigeminal nerve, thence to Meckel's ganglion, then through the large superior petrosal nerve to the geniculate ganglion, on through it and thence in the trunk of the facial to a point near its exit from the skull; there they leave in the chorda tympani nerve to join with the lingual branch of the fifth nerve and supply the sense of taste to the anterior two-thirds of the tongue (Fig. 49). It seems probable that in some cases the fibres for the sense of taste come from the glossopharyngeal through the nerve of Wrisberg and thus pass into the chorda tympani.

Secretory.—Fibres stimulating the secretion of saliva run in the nerve of Wrisberg, the geniculate ganglion and the chorda tympani to the corresponding side of the tongue, and when this nerve is irritated or paralyzed there are disturbances in salivatory secretion.

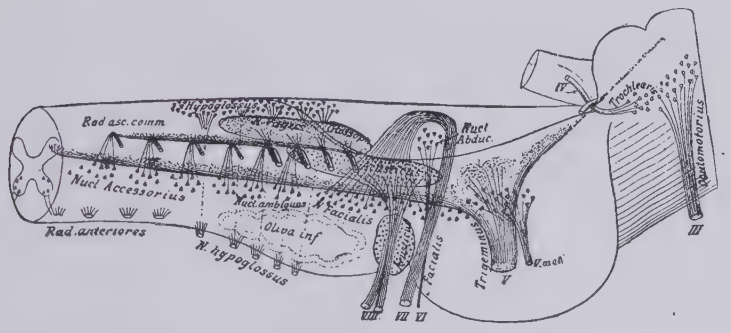


FIG. 48.—Longitudinal section showing position of the nuclei of medulla. (*Edinger.*)

Fibres which stimulate tear secretion pass in the seventh into the geniculate ganglion, thence through the great superior petrosal nerve, and thence to the lachrymal branch of the trigeminus.

The connections of the facial nerve with other nerves in its course through the aqueduct of Fallopius make it possible to determine the location of the different lesions which affect the facial nerve in this part.

The facial nerve being mainly motor, its diseases are spasmodic and paralytic. The two common types are facial tic and facial palsy, but there are other minor forms.

The spasmodic disorders are: (1) diffuse facial spasm or mimic tic and (2) spasm of single branches, including (*a*) blepharospasm and (*b*) nictitating spasm.

Facial Spasm (*Mimic Tic*).—This is a disease characterized by intermittent, involuntary twitchings of the facial muscles. It is always chronic and generally unilateral.

Etiology.—It is a disease of middle and later life, and occurs oftener in women; there is usually a neuropathic constitution; it is not hereditary. The exciting causes are shock, injury, and local irritations. It often has a reflex cause, usually from irritation of some branch of the

trigeminus. It is sometimes associated with tic douloureux. It may have an origin in reflex irritation of the sensory part of the seventh nerve, viz., the geniculate ganglion and nerve of Wrisberg. Organic diseases, such as tumors and softening, affecting the nerve nucleus in the pons or the cerebral centres, cause a symptomatic tic, but not the true disease. Thus we may have a post-hemiplegic tic or a tic due to cortical lesion and associated with epilepsy. A facial tic may also be caused by irritation or disease of the nerve trunk or of its nucleus. It sometimes, though rarely, follows a facial palsy.

Symptoms.—The disease usually begins slowly and the orbicularis muscle and zygomatici are earliest affected. It rarely goes above the

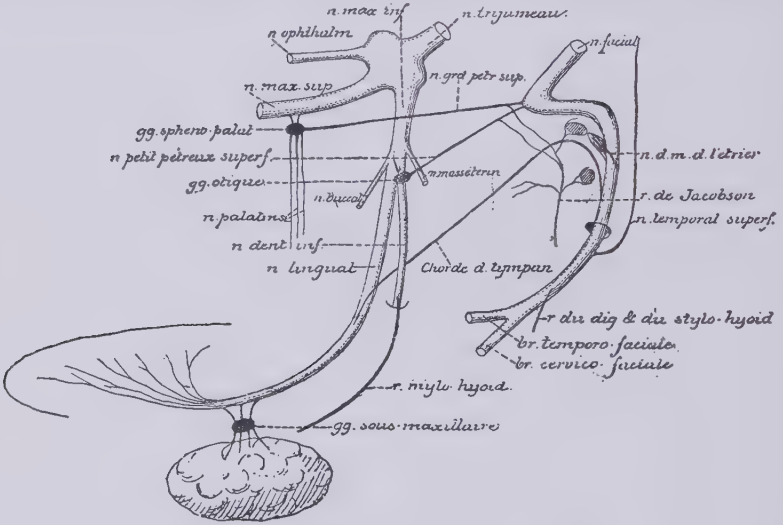


FIG. 49.—Showing the usual course of the taste fibres in the seventh and fifth nerves. (*Krause.*)

eyebrows, *i.e.*, to the corrugator supercilii and frontalis. The lower branch of the facial is little affected. The spasm is at first a clonic one; the muscles of the face are affected by a series of lightning-like twitches, with intervals of rest. Sometimes, however, the contraction becomes tonic and lasts several seconds or more. There is no pain. The spasm is increased by emotions, nervous excitement, conversation, exposure to light and cold, and is at its worst when the patient himself is most depressed. It is a very good gauge of the general nervous stability of the patient. There is no paralysis or atrophy and there are no secretory or trophic symptoms. The taste fibres are rarely involved, though occasional subjective sensations of taste have been felt. The electrical irritability is either unchanged or slightly increased.

Associated movements of the eyes, of the jaw muscles and cheek muscles are sometimes seen. Pressure over the motor points of the nerve will sometimes arrest the movements for a time. The disease is a unilateral one. It lasts for years and even for the lifetime.

Pathology.—There is no known anatomical change in idiopathic cases. The disease is allied in character to wry-neck and other chronic tics. In these cases it is probably the expression of some functional deterioration of the higher reflex centres. The disease is sometimes a pure reflex neurosis from ocular or dental or other irritations. In the facial tic that sometimes follows hemiplegia (nuclear tic) there is probably some degenerative change in the nucleus of the facial.

Diagnosis.—Idiopathic facial spasm is chronic, unilateral, unaccompanied by pain or paralysis. It is distinguished from facial spasms of organic origin by the fact that the latter always have some other symptoms. Thus facial habit chorea is bilateral; spasm from cortical disease is attended by disturbance of consciousness and comes on in paroxysms; the spasm occurring after hemiplegia is usually tonic, and so is hysterical facial spasm.

Prognosis.—The disease is in most cases incurable, especially after it has lasted some time. If a reflex cause exist, the prognosis is better. Life is, however, never endangered by it.

Treatment.—The most important thing is attention to the general health, removal of all depressing influences, rest, and freedom from excitement. Among specific remedies the bromides, parathyroid extract, the phosphates, gelsemium, conium, hyoscyamus, codeine and morphine are recommended. Morphine is useful, but must be tried carefully and in small doses. Conium lessens the spasm, but this drug has to be given in large doses and is not entirely free from danger. Hyoscine and gelsemium sometimes do good. Careful examination of the teeth, eyes, nose, stomach and uterus for reflex irritation is imperative.

Neurectomy of the supra-orbital, continuous pressure on the motor points, stretching the nerve itself are all measures which may be considered valueless. Freezing the skin over the nerve with chloride of methyl has been recommended by Mitchell. Blistering and cauterization are needless inflictions. The anæsthetization of the conjunctiva with cocaine is often helpful, both in diagnosis and treatment. Systematic training exercises, in which the patient sits before a mirror and tries to hold the spasm in control, and systematic facial and head movements help and may even cure these cases. I have seen a case apparently relieved by extract of parathyroid three times a day. Injections of alcohol into the nerve have cured some cases (Patrick).

Blepharospasm is the name given to a spasm, mainly tonic, of the

orbicularis palpebrarum. It is generally caused by diseases of the eye, and its nature and treatment are matters belonging to ophthalmology. It is a rare symptom of hysteria.

Nictitating or winking spasm is a clonic spasm of the orbicularis, and usually forms part of habit chorea or is a symptom of hysteria.

Tonic facial spasm is sometimes seen in major hysteria.

FACIAL PALSIES

The paralyses of the facial nerve may be due to lesions that are cerebral and supra-nuclear, or nuclear and peripheral.

Facial palsy of *supra-nuclear origin* is almost invariably an accompaniment of hemiplegia and is due to hemorrhage, softening, inflammation, or tumor of the brain. The lower two branches of the facial are chiefly involved.

Facial palsy of *nuclear origin* is very rare and is an accompaniment of glosso-labial palsy, of diphtheritic palsy, or of gross lesions of the pons.

Peripheral facial palsy (*Bell's palsy*) is the common type of facial paralysis, making up over 80 per cent. of cases. It is of infra-nuclear (peripheral) or nuclear origin.

Etiology.—The typical cases of this disease are due to exposure and so-called "rheumatic influences" with infection. After this the most frequent causes are ear disease, trauma, syphilis and tumors. Males are oftener affected, and the common age is between twenty and forty. It is more frequent in the winter and in temperate climates. It is not hereditary, but it may be congenital. A neuropathic tendency predisposes to it. Meningeal syphilis sometimes causes an isolated facial palsy, although it is apt to leave this nerve alone. Facial palsy may occur in multiple neuritis, when it is often bilateral. Non-typical and accidental cases of peripheral facial palsy are due to injuries, fracture of the petrous bone, or ear disease. Forceps pressure in difficult labor causes some cases. The ordinary cases are mostly due to microbic infections similar to those of "colds" and influenza.

Symptoms.—The disease comes on rather suddenly, and reaches its height within a few hours or, at most, two or three days. Preceding and accompanying the onset there may be some pain about the ears, and a little swelling is sometimes seen.

The patient feels a subjective discomfort on the paralyzed side of the face. He finds that he cannot completely shut the eye and if he tries to chew on the affected side, food gets between the teeth and cheek. He cannot pucker the lips, and his speech is a little muffled. The appearance of the face is most characteristic.

On the affected side the wrinkles are smoothed out, the angle of the mouth is lower, the mouth is drawn at first to the sound side, and owing to this distortion the tongue appears not to be protruded straight. The base of the tongue is lowered through paralysis of the stylo-hyoid and posterior belly of the digastric. In laughing or other emotional movements of the face, the trouble is most clearly brought out. But the most characterized appearance is produced by telling the patient to shut the eyes tightly and draw out the angles of the mouth so as to show the teeth (Fig. 50). The eye on the palsied side is not closed and the eyeball turns up, showing the white of the eye. In a forced attempt

Fig. 50.—Patient with left facial palsy trying to close both eyes and show the teeth.

at closing the eye, the upper lid may be drawn up. In children, and in the young and plump, the differences in the two sides of the face when at rest are not marked, especially in the slighter cases. The nostril on the affected side does not expand on forced inspiration; the eye is apt to be watery and the conjunctiva somewhat injected.

After a few weeks some wasting of the face may be noted, but this is never very great.

In some cases there is with the paralysis a great deal of pain in and about the ear and an herpetic eruption on the ear, which may be very slight in amount. A slight anæsthesia of the concha and posterior

wall of the external meatus is noted. In these cases, the trouble is due to an inflammation of the geniculate ganglion which has extended and affected the facial nerve and the primary trouble is an herpetic inflammation of the ganglion (J. R. Hunt).

The electrical reactions are characteristic and important, since typical or partial degeneration reactions can usually be observed. For a few days there is an increase in irritability of the muscles to both faradic and galvanic currents. This is followed by a gradual loss or diminution in faradic irritability, while the galvanic irritability remains sometimes increased for a while and then falls. At the same time a reversal of poles and a sluggish contraction, particularly the latter, may be noted. At the end of five or six weeks faradic irritability ought to begin to return. Great variations are found in the electrical reactions, dependent upon the severity of the case. For example, in very severe cases the electrical irritability may be almost or entirely absent for days and even weeks.

If the disease lasts for two months or more and the palsy is not a complete one, secondary contractures begin to appear. The mouth now becomes drawn to the affected side, and the nasolabial fold becomes deeper than that on the sound side. In smiling or speaking or other facial movements there is an excess of movement on the paralyzed side, the teeth and upper gums in particular showing abnormally. This is particularly the case in old palsies beginning in childhood.

Pathology.—The disease in its typical form is due to a diffuse neuritis. This attacks the periphery of the nerve in the face and extends rapidly up into the Fallopian canal as far as the geniculate ganglion. The inflammation sometimes attacks most the peripheral filaments; at other times it is more central.

Clark has recently brought evidence to show that the old idea of a "Fallopian neuritis," pinching the nerve and cutting it off in the canal, is the correct one.

As already stated, Dr. Hunt has shown that some cases of facial palsy are due to the extension of an inflammation of the geniculate ganglion, which is the sensory ganglion of the seventh nerve.

Bell's palsy is not a "rheumatic" disease; it is an infection, and should be classed as such. Many cases occur after influenza or some malady allied to it. A long list of acute infections which are occasionally followed by Bell's palsy may be collected, but in the great majority of cases it is much like a "cold" of the seventh nerve.

This view is borne out by experience in treatment which shows that antirheumatic drugs, like the salicylates and iodides, do not appreciably modify its course.

Diagnosis.—The recognition of the palsy is made easy by causing the patient to contort the face. In children it requires more care to detect the side affected.

It is important to determine whether the palsy is cerebral, nuclear, basilar, or peripheral.

There is a difference in the grouping of symptoms in accordance with the location of the lesion.

1. When the lesion is supra-nuclear, that is between the cortex and the nucleus of the seventh, the paralysis of the face involves only the lower or cervico-facial branches, and the patient is able to close the eyes, elevate the eyebrows and wrinkle the forehead. There are no electrical reactions of degeneration.

2. When the lesion is in the nucleus itself, there is very apt to be an associated paralysis of the sixth nerve on account of its close anatomical relations. Taste and hearing are unaffected.

3. When the lesion is at the exit of the nerve from the pons, central to the geniculate ganglion, there is a typical facial paralysis without affection of taste; but as such a lesion implicates usually the auditory nerve, there is usually deafness. The stapedius is paralyzed, and if the auditory nerve is injured some disturbance in hearing may result, and there may be disturbance of tear secretion on the paralyzed side.

4. If the lesion is within the aqueduct of Fallopius between the geniculate ganglion and the point where the chorda tympani leaves the nerve, there is loss of taste on the anterior two-thirds of the tongue on the affected side and sometimes disturbance in the secretion of saliva. This may cause a furring of the tongue on this side.

5. If the lesion is far enough up in the canal to involve the stapedius, there is sometimes great sensitiveness to loud sounds, or sometimes to low sounds. (This symptom is much rarer in the breach than in the observance.) The patient cannot produce the peculiar sound caused by blowing out the cheek and forcing air into the ear.

6. If the lesion affects the facial nerve at a point peripherally to its exit from the stylo-hyoid foramen, we have a complete paralysis of the muscles of the face, but there are none of the disturbances of secretion or taste or lachrymation or hearing.

Practically Bell's palsy of the ordinary type involves portions 4, 5, or 6. If central, the disease is usually of syphilitic or tuberculous origin; the palsy is severe and the loss of ability to close the eye very great. In the geniculate ganglion cases there is usually herpes, great pain and a slight cutaneous anæsthesia, as described. (Hunt's Syndrome.)

Prognosis.—The prognosis of peripheral palsy (Bell's) is good, although an absolutely complete recovery often does not take place.

In syphilitic cases the prognosis is not so good, though patients may recover. In supra-nuclear (central) palsies the prognosis is the worst, because the lesion (hemorrhage, softening, tumor, etc.) usually does not disappear.

However, the central cases are from the beginning of a mild type, and give annoyance mainly from the secondary contractures.

Bell's palsy usually lasts three to five months. Occasionally there are mild cases that get well within a month. The prognosis as to duration is much helped by a close study of the electrical reactions. In proportion as the degeneration reaction is complete and persistent, the outlook is bad.

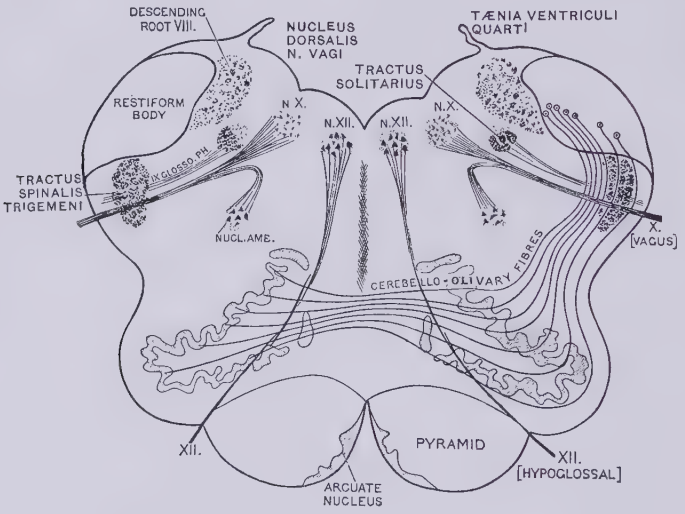


FIG. 51.—Section of medulla showing origin of ninth, tenth and twelfth cranial nerves. (*Cunningham.*)

Treatment.—In the acute peripheral cases the patient should be treated promptly and thoroughly. He should be given a diuretic and purgative and a blister should be placed over the exit of the nerve. This should be followed or accompanied by hot fomentations. Salicylate of soda in full doses of 20 grains may be given during the first week. After the paralysis is established, iodide of potassium is indicated in moderate doses. Electricity is to be employed with care at first. After a week it may be given daily for five minutes, using the galvanic currents just strong enough to contract the muscles. After three or four weeks, if the faradic current causes contraction it may be used, otherwise the galvanic current is to be continued.

At the end of three weeks, if the paralysis is severe, the corner of the *mouth should be drawn up by means of a bent hook,* which is carried back

and fastened behind the ear. The patient should wear this most of the daytime, but not at night. The object is to take off the strain caused by the pulling of the muscles on the sound side.

At the end of a couple of weeks massage should be given, and the patient should practice facial gymnastics daily. If the eye cannot be closed it should be protected by a shade. In obstinate cases good results have been obtained by suturing part of the hypoglossal nerve to the facial.

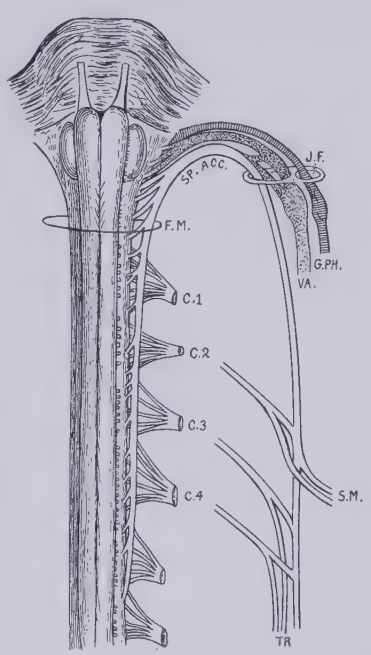


FIG. 52.—Vagus and spinal accessory nerves. (*Cunningham.*)

THE GLOSSOPHARYNGEAL NERVE

Anatomy.—The glossopharyngeal nerve has motor, sensory and autonomic fibres. Its motor function is very slight, supplying only the stylo-pharyngeus.

The nucleus of origin of the motor fibres is the nucleus ambiguus common to it and the vagus and accessory. The sensory fibres arise from two small ganglia lying on the root of the nerve, the *petrous* and *jugular*.

The nerve supplies general sensation to the tympanum, tonsils and pharynx (in connection with the vagus) and upper part of the larynx; special sensation of taste to the posterior third of the tongue, and motion to the pharyngeal muscles and œsophagus (Kriedl) in connection with the vagus.

The terminal filaments of the sensory taste fibres supplying the posterior two-thirds of the tongue end in fine fibres that pass into the taste buds. There are no special peripheral cells of taste, as asserted by Fusair and Panasci.

Its cortical representation so far as taste is concerned is in the hippocampal gyrus. The nerve gives very sensitive reflex fibres to the pharynx and is important in the reflex act of deglutition; it also carries sensations of nausea from pharyngeal irritation.

MOTOR NEUROSES OF THE GLOSSOPHARYNGEAL

This nerve has very little motor function and is rarely affected independently by motor troubles. Spasm of the pharyngeal constrictors occurs in general disorders like rabies, and reflexly in severe neuralgia of the trigeminus. This condition, known as *dysphagia*, is seen also in hysteria, and there is probably sometimes spasm in connection with the symptom known as globus hystericus.

Paralysis of the throat constrictors occurs as one of the symptoms of glosso-labio-laryngeal paralysis and sometimes in diphtheritic paralyses.

THE PNEUMOGASTRIC NERVE AND THE ACCESSORY PART OF THE SPINAL ACCESSORY

Anatomy.—The pneumogastric or vagus nerve belongs to the autonomic nervous system. It has two nuclei of origin, a motor and a sensory.

1. The motor nucleus or nucleus ambiguus, which is a prolongation of the lateral horn of the spinal cord lies deep in the medulla and has a nucleus common to the vagus and glossopharyngeal (Fig. 51).

2. The sensory fibres arise chiefly from two ganglia that lie on the root of the nerve, the jugular and plexiform. These bodies resemble spinal ganglia. The cells are unipolar and send off a neuraxon which bifurcates. The peripheral fibre passes on along the nerve and supplies it with its sensory fibres. The central part passes up to the gray matter of the floor of the fourth ventricle and ends in the so-called sensory nucleus (Fig. 47). This nucleus, however, is not the real nucleus of origin, but, as in the case of the ninth, it contains cells which send their axis-cylinders or neuraxons brainward and form secondary sensory neurons. Fibres also go to the ascending sensory root common to this nerve and the ninth.

The spinal accessory nerve is purely motor in function (Van Gehuchten). The accessory part arises from the cells of the nucleus ambiguus and passes into the trunk of the vagus.

The spinal part of the spinal accessory arises from the lateral horn and outer part of the anterior horn of the spinal cord. Its fibres of orign reach from the first to the third or fourth cervical nerves and as far as the fourth or fifth cervical roots. The fibres unite in the cranium and pass out through the posterior lacerated foramen in the same sheaths as the vagus. After their exit from the skull, they divide into an internal and external part. It is the external branch which contains the fibres of spinal origin. The internal part contains the fibres from the medulla and unites with the vagus. The nerve receives some sensory fibres from the first and sometimes from the second cervical nerve. The terminal branches receive motor fibres from several cervical nerves. The spinal accessory contains large and small or visceral fibres. The spinal part contains only the large fibres.

The spinal accessory supplies the sterno-cleido-mastoid almost exclusively, but only the upper part of the trapezius; the rest of this muscle is supplied by the cervical and dorsal nerves. The sterno-cleido-mastoid, when innervated, draws the chin up and over toward the opposite side. The upper fibres of the trapezius draw the head back slightly and down toward the same side. Physiologically, the spinal part of the accessory nerve is one of the motor cervical nerves; the accessory or medullary portion is part of the vagus, and has visceral and sensory as well as motor functions.[1]

The vagus and accessory part of the eleventh together have an extraordinary wide distribution and diversity of function.

1. First they contain motor, inhibitory, and vasomotor fibres. These fibres go to the pharynx, larynx, trachea and bronchi, to the œsophagus, stomach, small intestines and spleen.

2. Sensory fibres, which go the occipital and transverse sinuses and dura mater of the posterior fossa, to the external auditory meatus in part, to the pharynx, larynx and trachea and to the œsophagus.

3. Excito-reflex fibres, which go to the lungs and heart, stomach, and to other organs mentioned as supplied by the vagus with sensation.

These reflex fibres stimulate or inhibit the vasomotor centre, the respiratory rhythm and the cardiac rhythm. They also excite reflexly deglutition and respiratory movements.

[1] The special autonomic character of the vagus has been noted under the head of the sympathetic nervous system.

The secretory fibres go to the respiratory tract, œsophagus, stomach and pancreas and small intestines.

Cardio-inhibitory fibres go to the heart, while reflex accelerating fibres and inhibiting fibres go to the lungs. The accessory nucleus supplies the laryngeal adductors and the cardio-inhibitory fibres.

DISEASES OF THE VAGUS—ACCESORY NERVES—VAGOTONIA

These nerves are essentially autonomic in character. Their diseases call for a study of laryngeal, pulmonary, cardiac and abdominal neuroses, which would bring us into the domain of laryngology and general medicine. Hence, despite their great importance, I have thought it best to describe only a special neurosis due to functional vagus disturbance and in a general way, the anatomical supply and function of the laryngeal nerves. Some of the other symptoms of its disorders are described in connection with locomotor ataxia, progressive muscular atrophy, exophthalmic goitre, and angina pectoris.

VAGOTONIA

There has been, however, an attempt of late to show that sometimes the pneumogastric nerve is thrown into acute tonus or stimulation, often in association with other cerebral autonomic nerves. This condition is called vagotonia.

Eppinger, Hess and others have worked out a neurosis of the cerebral autonomic system.

In a typical vagotonic attack all or nearly all of the cerebral autonomic nerves are affected. As the result, there is narrowing of the pupil from irritation of the ciliary ganglion (III nerve), salivation and flow of tears, from irritation of the chorda tympani and lachrymal nerve (VII nerve), hyperidrosis from irritation of the cervical sympathetic, bradycardia and respiratory arythmia, bronchial asthma, hyperacidity and increased gastric motility, from irritation of the vagus.

This group of symptoms has this in common, that all are alleviated by large doses of atropia, and all are increased by pilocarpin. It is a clinical picture built up more on pharmacological than clinical data. I think we see only the fragments of it.

A commoner form of vagotonia, it seems to me, shows itself as a real clinical phenomenon much more frequently in what Gowers has described as vagal and vaso-vagal attacks. These are characterized by subjective or sensory disturbances mainly. The patient, for example, first feels a sensation of distress or distention of the stomach, which may be associated even with nausea and faintness. With it there comes a feeling of respiratory distress and inability to get the breath, a feeling of impending death or a sensation of fainting. There is generally a rather slow pulse and cold extremities. Occasionally the patient feels a very heavy beat-

ing of the heart. There is flatulence, and when gas is raised the symptoms are relieved in part. The stomach shows increased acidity and evidence of excessive motility or spasm.

There is a mental condition of confusion and alarm, and this often simulates hysteria, and these vagal seizures are often regarded as hysterical.

The Laryngeal Nerve-supply.—Phonation is mostly a voluntary act, and has a centre in the cortex of both hemispheres at the lowest level of the præcentral convolution. By its stimulation adduction of the vocal chords can be produced.

Respiratory movement (abduction of the vocal chords) has no cortical centre. Its centre is in the medulla near the vagal nuclei.

The motor nerve of the larynx is the recurrent laryngeal which innervates all the muscles, adductor as well as abductor, except the cricothyroid which is innervated by the superior laryngeal.

The motor fibres for the abductors (respiratory fibres) run in a separate bundle inside the recurrent nerve while the adductor or phonatory fibres run on the outer part (R. Russell).

The abductors are paralyzed first and most easily and are especially apt to be affected by progressive organic disease in the bulb.

But in functional diseases (psychic, hysterical) the adductors are exclusively affected. This is because the impulses to the adductor muscles have a wider range of fibres to work upon. When there is complete recurrent nerve palsy, the vocal chord is motionless and in a position near the median line.

The superior laryngeal is the sensory nerve of the larynx, and when it is injured there is anæsthesia of the larynx and paralysis of the cricothyroid which is a tensor muscle.

In vagus paralysis high up on one side we have anæsthesia of the larynx with loss of reflex phenomena and consequent passage of foreign bodies, food, etc., into the larynx; also tachycardia and paralysis of the abductors, which may be bilateral.

In paralysis of the superior laryngeal we have only the anæsthesia of the larynx with its resulting loss of reflex protective activity.

In paralysis of the recurrent laryngeal we may have at first and only abductor paralysis, later total paralysis of the vocal cord.

The causes are injuries, infectious and degenerative diseases, such as tabes, syphilis, progressive bulbar palsy, and any organic bulbar lesions, such as tumors, hemorrhage and softenings. The recurrent laryngeal suffers sometimes from what is probably an infectious neuritis, causing subacute paralysis of one abductor which is recoverable. The commonly observed cause is aneurism of the aorta and tumors of the mediastinum or neck, such as goitre.

NEUROSES OF THE SPINAL PART OF THE ACCESSORIUS AND UPPER CERVICAL NERVES

The accessory is a purely motor nerve, and its disorders are therefore spasm and paralysis.

Torticollis (*Wry-neck, Caput Obstipum*).—Torticollis is a disease characterized by clonic or tonic spasm of the muscles supplied by the spinal accessory and often of other muscles of the neck. There are several forms of wry-neck, which must be distinguished from each other. They are: (1) congenital wry-neck; (2) symptomatic wry-neck; (3) spurious wry-neck, from spinal disease; (4) true spasmodic wry-neck.

1. *Congenital wry-neck* is due to some intra-uterine atrophy or obstetrical injury of the sterno-cleido-mastoid. It occurs oftenest after

FIG. 53.—Congenital wryneck of the right side.

breech or foot presentations. The right side is usually affected. There is no spasm at all, but the neck is fixed to one side by the shortness of the muscle, and also rotated to the opposite side. The deformity becomes more noticeable as the child grows older, because the parts atrophy. The atrophy affects not only the shortened muscles, but the face on the affected side (Fig. 53).

2. *Symptomatic wry-neck* is usually due to a rheumatic myositis, and occurs chiefly in children. It may be due also to tumors, adenitis, abscesses and local syphilitic disease. In these cases there are always pain and tenderness associated with the deformity.

3. *Spurious wry-neck* is an apparent or real spasm of the neck muscles due to caries of the spine. A spurious wry-neck may be caused by defect of the oculo-motor apparatus such as lateral nystagmus (Starr).

Treatment.—Congenital wry-neck, if taken early, can be cured by tenotomy of the sterno-mastoid and subsequent fixation of the neck

for a time. When osseous changes have occurred perfect relief is impossible. Symptomatic rheumatic wry-neck is a trivial and temporary affection, which needs only to be palliated by hot applications, salicylates and saline purges until cure takes place.

Spurious wry-neck requires suitable orthopædic measures, such as the plaster jacket and jurymast.

4. **Spasmodic Wry-neck.**—This is a purely nervous disease characterized by spasm of the muscles supplied by the spinal accessory and generally of those supplied by the upper cervical nerves also.

Etiology.—Women are oftener affected than men. It arises in early adult and middle life, never in children or old people. The sufferers

Fig. 54.—Typical wryneck involving the spinal accessory on the right side. (*Walton.*)

are of a neuropathic constitution and direct heredity may exist. Overwork and exhausting emotions are apparent predisposing causes.

The exciting causes are occupations which put the lateral muscles of the neck on a strain, or in which frequent lateral movements of the head have to be made. It is then an occupational neurosis like writer's cramp.

Trauma, peripheral irritation, eye-disease, various infections and toxæmic states which depress, may excite the disease in a neuropathic person.

Symptoms.—The disease begins with slightly painful sensations in the neck, which are soon accompanied by spasm. The spasm is at first clonic and intermittent. The sterno-mastoid is oftenest involved of single muscles, but the rule is that the upper fibres of the trapezius are also affected. The patient's head is inclined toward the affected side by the trapezius, the chin is raised, and the head rotated to the opposite side by the sterno-mastoid and trapezius, and this is the typical position in the disease (Fig. 54). If both trapezii are affected the head

is pulled back, but this is a rare form. It is called retro-collic spasm. The complexus and obliquus superior are the only other neck muscles which can rotate the head to the opposite side. They are supplied by the upper cervical nerves and are often involved in wry-neck. In torticollis the muscles affected with spasm have a similar physiological function. Hence, while the sterno-mastoid, trapezius, complexus and superior oblique on one side are attacked by the spasms, muscles on the other side may be at the same time implicated. The opposite muscle commonly affected is the splenius, which inclines the head laterally and rotates it to the same side. Probably the deep muscles, recti capitis postici, major and minor, and the inferior oblique, which draw the head back and rotate to the same side, are also at times affected. The list of muscles that may be involved and their nerve supply are as follows:

	Turning head to Opposite Side	Turning or Inclining Head to Same Side	Nerve Supply
Muscles usually involved	Sterno-cleido-mastoid....	.....	Eleventh.
	Upper fibres of trapezius.	.....	Eleventh
Muscles rarely involved	Superior obliquus.....	Recti capitis postici, maj., min., infer. obliq., splenius.	Cervical.
	Complexus.....		Cervical.

Extreme rotation without much retraction of the head would indicate involvement of the sterno-cleido-mastoid and opposite splenius. Retraction of the head indicates involvement of both trapezii or the deep recti muscles. A full account of the physiology of the neck muscles is given later under the motor neuroses of the upper cervical nerves—where torticollis more properly belongs.

The disease may start in one muscle and gradually extend to others, even involving the facial, masticatory and brachial nerves. As it progresses the spasm becomes more constant, and finally it may be tonic, never yielding except to artificial means or during sleep. The pain associated with the disease gradually decreases. The affected muscles hypertrophy, the muscles thrown into disuse atrophy. There is some deformity, in time, of the neck and shoulders, but facial asymmetry does not occur in this form unless it begins, as is very rarely the case, before maturity.

The disease reaches a chronic stage in one-half a year to a year, then remains stationary or gradually improves. The disease may be complicated with other neuroses or a psychosis.

Pathology.—Torticollis is a psycho-neurosis involving the bulbar and lower psychic centres. The disease is in its usual form a degenerative

one, and indicates the premature decay or exhaustion of centres never perhaps originally perfect. It is called in its typical form by French writers "mental torticollis." But all true torticollis is "mental," in the sense that the normal volitional control is lost and the psychic part of the mechanism is disturbed. It is not "mental" in the sense that it is dependent on an idea, except in cases occurring in early life when it may be psychogenic.

The *diagnosis* has to be made from the other forms of wry-neck mentioned. The age, history and fixed character of the spasm serve to distinguish congenital wry-neck. The history, the pain and tenderness and the temporary duration differentiate the rheumatic forms.

The increased rigidity on passive motion, the pain, deformity, and other signs of cervical caries are sufficient to diagnosticate vertebral disease.

Prognosis.—The disease is not fatal. It generally reaches a certain stage and then remains chronic. In young and psychogenic cases it is cured; in many others it can be much ameliorated.

Treatment.—The drugs which are useful are very few. Opium, chloral, atropin, the bromides, and zinc are generally the most efficacious. Opium must be given with great caution. Atropin should be given hypodermically in increasing doses up to intoxication (gr. $\frac{1}{10}$) (Leszynsky). The galvanic and faradic currents are useful adjuvants in helping to relax the spasm and keep up the nutrition of the muscles, but alone they are of little value. Massage and stretching the neck in a Sayre apparatus, together with systematic exercise of the neck muscles, often help. The only surgical measures to be advised are nerve resection, and possibly the partial cutting of the sterno-mastoid muscle.

The *pedagogical and psycho-therapeutic method* of dealing with torticollis, by rest, massage, exercises and relaxation, is the most important of all measures. This form of treatment must be combined with rest, tonics and symptomatic remedies, and it must be begun if possible early and kept at persistently and repeatedly—if necessary even for two or three years. Prolonged rest for months or even much longer with pedagogical, local and general exercises are the basis of rational treatment and often of practical cure.

Psycho-analysis and hypnotism do no good, except perhaps in young and distinctly hysterical patients.

Splints and mechanical-fixation apparatus do no good, as a rule. Alcoholic injections may be tried. I have never seen permanent good from surgery.

Paralysis of the Spinal Part of the Accessory—*Etiology.*—The chief causes are injuries, caries of vertebra, progressive muscular atrophy, and anterior poliomyelitis.

Symptoms.—When one nerve is paralyzed the head may still be held straight, but there is inability to rotate it perfectly. The prominence of the sterno-mastoid is absent—atrophy takes place. No spasm of the other muscle occurs, and there is no such thing as paralytic torticollis (Gowers). The involvement of the trapezius causes a depression in the contour of the neck, especially noticeable on deep inspiration. There is some trouble in raising the arm, the scapula is drawn away from the spine, and the lower angle is rotated inward. When both nerves are paralyzed there is great difficulty in rotating the head or raising the chin. Paralysis of both sterno-mastoids causes the chin to drop backward, while paralysis of both trapezii in their upper parts causes the head to drop forward. Atrophy of the muscles attends the paralysis of the nerve, and degenerative reactions may be noted. The cervical nerves appear sometimes to supply the sterno-mastoid and upper part of the trapezius so much that in disease of the accessories decided paralytic symptoms may be absent.

The *diagnosis* depends upon a thorough examination of the motility of the parts.

The *treatment* is based on a knowledge of the cause of the disease.

THE HYPOGLOSSUS—XII

Anatomy.—The hypoglossal nerve arises from a long and large nucleus lying on the lower part of the floor of the medulla near the median line and to the outer and ventral side of the central canal. The nucleus is a continuation upward of the anterior horns of the spinal cord and is homologous with the sixth, fourth and third nerve nuclei higher up (see Fig. 47). It reaches below as far as the decussation of the pyramids and above as far as the glosso-pharyngeal nucleus. A second small-celled nucleus lies just beneath the nucleus proper. Its cortical representation is in the lower end of the præcentral convolutions, to which it is connected by fibres that pass into the raphe and thence to the anterior pyramids. Its fibres pass out between the olivary body and the anterior pyramid. At its origin it is a purely motor nerve; it receives a few sensory fibres from the cervical nerves and the vagus. It supplies the intrinsic and extrinsic muscles of the tongue: It is also thought to send fibres to the oral muscles (Tooth).

The hypoglossal nerve is concerned in the movements of the tongue and in fixing or depressing the hyoid in mastication and deglutition. When diseased, therefore, speech and deglutition are affected. The small nucleus of the nerve is thought to control the finer lingual movements of articulation.

MOTOR NEUROSES OF THE HYPOGLOSSAL NERVE—THE MECHANISM OF ARTICULATION

The diseases of this nerve consist of lingual spasms, lingual palsy and lingual hemiatrophy.

Lingual spasms take part in the disorders of articulation, helping to cause stuttering and speech cramps. Such troubles are often developmental in origin and belong to the habit choreas or convulsive tics.

Stuttering is a spasmodic disorder of the mechanism of articulation, causing a habitual and spasmodic repetition of words, or syllables.

Stammering is spasmodic abortive speech caused, according to E. Thompson, by the effort of the stammerer consciously to do what should, and must be done, automatically.

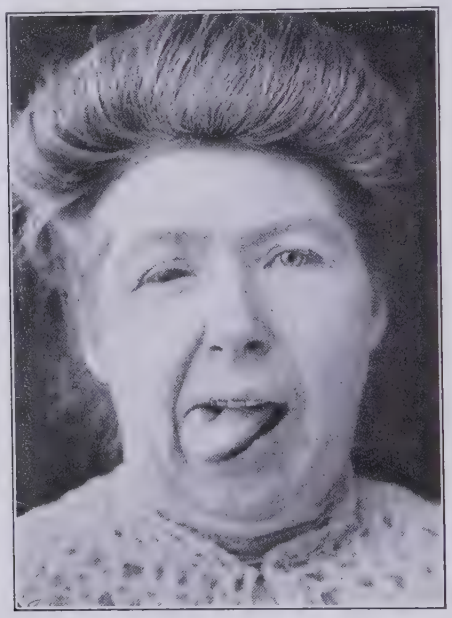


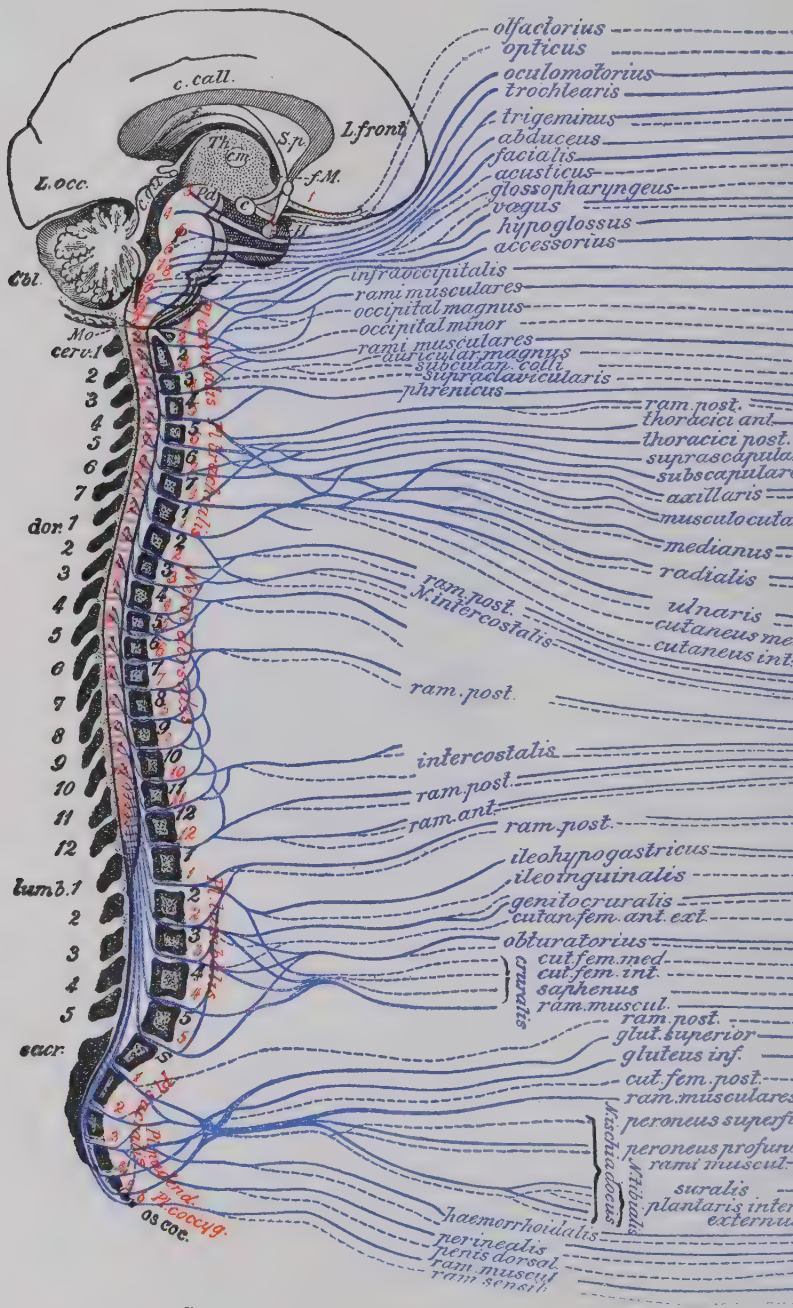
Fig. 55.—Left lingual hemiatrophy.

Aphthongia is the name given to a form of spasm occurring in speakers and similar in nature to writer's cramp.

Clonic lingual spasm occurs in chorea, hysteria and during the attacks of epilepsy. Unique cases of this spasm also occur from reflex irritation or central nervous disease.

Tonic lingual spasm occurs in hysteria, and sometimes as an independent affection due to local, toxic or reflex irritation.

Lingual paralysis (*glossoplegia*) is usually one of the symptoms of glosso-labio-laryngeal palsy. It may also be caused by a bilateral or even a single lesion in the cerebral hemispheres. The condition is then known as pseudobulbar paralysis. Diseases of the medulla and of the nerve itself may cause paralysis and atrophy. The paralysis may be either unilateral or bilateral. The symptoms are an impairment of



SCHEME OF NERVE DISTRIBUTION. (*Jakob.*)

"The segments are printed in red. The motor nerves or portions of mixed nerves in solid blue lines. The sensory nerves or sensory portions of mixed nerves in dotted blue lines."

(a) interior eye muscles (intercalation of the ciliary ganglion), ciliary muscle for accommodation —sphincter of the pupil for contraction of the pupil.
(b) exterior eye muscles; levator palpebræ superioris; rectus superior, internal, inferior— inferior oblique, movement of the eyeballs upward, inward, and downward.

Superior oblique of the eye (movement downward and outward).
Muscles of mastication; masseter, temporal, pterygoid, mylo-hyoid—ant. digastric, tensor tympani, tensor and levator veli palati. Secretion of tears?

External rectus of the eye (outward movement).
The mimic face muscles; frontal, orbicular of the eye and mouth; zygomatici, mental; platysma. Of the ears, stapedius, posterior digastric, etc. Nasal secretions. Tear secretion? Palate musculature?
Throat musculature (constrictors), crico-thyroid, secretion of gastric juice. Movements of heart
Tongue musculature, sterno-hyoid, thyreoidei. [and breathing.
Larynx musculature (voice formation), palate, pharyngeal musculature (with the vagus) (act of swallowing)—sterno cleido-mastoid (drawing the head laterally and nodding). Trapezius (in part) shrugging the shoulders).

Posterior superficial and deep head and neck muscles (elevating and lateral movements of head).
Deep posterior and anterior back muscles. Trapezius in part (movement of head and throat).

Scaleni (elevators of the ribs, breathing), longus colli.

Diaphragm (breathing).

Deep neck muscles.
Pectoralis major (anterior adduction of the upper arm), pectoralis minor, subclavius.
Levator scapulæ, rhomboids (dorsal nerves of the scapula) (draws the scapula inward and upward), serratus anticus major (long thoracic nerve) fixes the shoulder blade and draws the same in conjunction with the arm above the horizontal.

Supraspinatus (raises and rolls the arm outward), infraspinatus, teres minor (rolls outward).
Subscapularis, teres major (rotates inward), latissimus dorsi (adducts the arm and draws it backward).
Deltoid (raises the arm to horizontal).
Biceps (flexor of forearm and supinator), brachialis anticus (flexor of forearm), coracobrachialis.
Flexor communis digitorum (radial portion) (bends the terminal phalanges), flexor longus pollicis (end phalanx), flexor carpi radialis, pronator radii teres, and quadratus, palmaris longus, thenar muscles (opponents of thumb flex the primary phalanges and extend the terminal phalanges), lumbricales 1, 2, seldom 3 (flex the peripheral phalanges and extend simultaneously the terminal phalanges).
Triceps (extensor of forearm), radial portion of brachialis anticus, supinator longus! (flexes and pronates the forearm), extensor communis digitorum (extensor of the peripheral phalanges), extensor pollicis—abductor pollicis, supinator brevis, extensor carpi radialis and ulnaris.
Flexor profundus digitorum (ulnar portion), see 20. Flexor carpi ulnaris, hypothenar, interossei (flex the peripheral phalanx), lumbricales (3 and 4), see 20, adductor pollicis.

Deep back muscles.

Intercostal muscles.

Deep back muscles (extensor of trunk).

Intercostal muscles.

Back muscles.
Intercostal muscles, abdominal muscles (rectus, external oblique) (compressor of abdomen).

Back (lumbar) muscles.

Transverse abdominal, internal oblique (compressor of abdomen).

Cremaster, transverse, oblique.

External obturator, adductor of thigh, gracilis (adductor).

Psoas (lumbar plexus), iliacus internus (draws up the thigh, flexes the trunk), quadriceps (extends the leg), sartorius.
Gluteus medius, minimus (abductors of thigh), tensor vaginæ femoris, pyriformis, obturator internus (abductors).
Gluteus maximus (extends the thigh).
Gemelli; biceps, semitendinosus, semimembranosus (flex the leg), quadriceps femoris (adductor).

Tibialis anticus (raises the inner border of foot), peronei (raise outer border of foot), extensor digiti communis.
Gastrocnemius, soleus (plantar flexion of the foot), flexor digiti, tibialis posticus.

Small muscles of foot (flexor brevis, interossei, etc.).
Levator, internal sphincter ani, sphincter vesicæ.
Sphincter ani, perineal musculature, bulbi cavernosi, etc.
External sphincter ani.

speech and of swallowing. Fuller details will be given under the head of Bulbar Palsy.

Progressive Lingual Hemiatrophy.—A progressive hemiatrophy of the tongue sometimes occurs. It is analogous in all respects to facial hemiatrophy, with which it is sometimes associated. It is probably due to a low grade of degenerative neuritis of the nerve.

CHAPTER IX

NEUROSES OF THE MOTOR SPINAL NERVES

Anatomy and Physiology.—The spinal nerves arise from the spinal cord by two roots, anterior and posterior. These roots unite outside the spinal canal to form mixed nerves. The mixed nerves divide and go to their various destinations. There are thirty-three pairs of spinal nerves, viz.:

Cervical	8	
Dorsal	12	
Lumbar	5	
Sacral	5	
Coccygeal	3	(all rudimentary).
	33	

The last two coccygeal nerves are microscopic in size, and the first pair is very small, so that practically there are but thirty sets of spinal nerves.

The posterior roots are closely connected with ganglia lying in the intevertebral canal, and called intervertebral gangla, or ganglia of the posterior roots. These ganglia are the real origin of the great majority of the fibres of these roots. The mixed nerve is connected by fibres that come chiefly from the anterior root, with the sympathetic or prævertebral ganglia. The distribution of the spinal nerves is shown in Plate III.

For the purpose of conveniently studying the diseases of the spinal nerves, we divide them into six different groups, each having a somewhat definite work to do. These groups are shown in the accompanying table.

	Strands of Spinal Nerves	Distribution	Associated Ganglia of Sympathetic
Group I.	Upper four cervical.	Occipital region, neck.	First cervical.
Group II.	Lower four cervical and first dorsal.	Upper extremities.	Second and third cervical, first dorsal.
Group III.	Upper six dorsal.	Thoracic wall.	First to sixth dorsal.
Group IV.	Lower six dorsal, except last.	Abdominal wall, upper lumbar, upper lateral thigh surface.	Fifth to twelfth dorsal.
Group V.	Twelfth dorsal, four lumbar.	Lumbar region, upper gluteal, anterior and inner thigh and knee.	First to fourth lumbar.
Group VI.	Fifth lumbar and five sacral.	Lower gluteal, posterior thigh, leg, pelvic organs.	First to fifth sacral.

GROUP I. THE UPPER CERVICAL

includes the first four of the spinal nerves. These divide into anterior and posterior branches. The posterior branches supply the muscles and skin of the back of the

neck and the occiput. The principal nerves are the suboccipital and the great occipital.

The anterior branches form the cervical plexus. Its principal branches are the auricularis magnus, occipitals minor, and phrenic. The special distribution of the nerves is shown in the table. (See Appendix.)

The upper cervical nerves supply motion to the muscles which rotate the head and draw it back and sideways.

One branch, the phrenic, supplies the diaphragm; others supply muscles which assist in fixing the thorax in forced inspiration. They innervate some of the hyoid and thyroid muscles, but have no influence on phonation or deglutition. This group of nerves is in close connection centrally with the trigeminal nerve, whose descending root reaches down into the cervical cord. The fibres to the scalp and face also anastomose with the trigeminus in their peripheral distribution to the scalp and chin.

Special Anatomy.—A knowledge of the anatomy and physiology of the neck muscles and nerves is important in studying the more serious neuroses of this region.

Muscles	Function	Nerve Supply
The platysma myoid...	Wrinkles the neck, depresses the jaw and angle of the mouth.	Facial.
Recti capitis antici....	Flexion of head.	C^1 and C^2.
Recti capitis laterales..	Lateral movement and slight rotation.	Antr. branches, $C^{1,2,6}$.?
Scaleni med. and post..	Lateral movement and slight extension.	Antr. branches, C^6,?
Sterno-mastoid.....	Lateral movement; also flexion and rotation of head to opposite side.	Spinal accessory and C^2
Trapezius.....	Lateral movement and extension.	Spinal accessory and anterior branches of C^3 and C^4.
Levator anguli scapuli.	Lateral movement and rotation to same side.	Anterior branches, C^3 and C^4.
Splenius capitis.....	Draws head backward.	Ext. div. of post., branches of cerv. nerves. C^2, C^3.
Trachelomastoid.....	Draws head backward, turns face to same side?	Post. branches cerv. nerve, C^3.
Complexus.....	Draws head back or rotates to same side.	Post. branches cerv. nerve C^1, C^2, C^3.
Obliquus superios.....	Extends and slightly rotates to opposite side and bends to same side.	Post branches cerv. nerve C^1.
Obliquus inferior.....	Rotates to same side.	Post. branches cerv. nerve, C^2.
Rectus cap. post.....	Extends and rotates to same side.	Post. branches cerv. nerve. C^1.

The movements of *flexion* of the head are thus seen to be due to the sterno-mastoid and anterior recti capitis, innervated by the spinal accessory and by the C^1 and C^2 anterior branches). In forced flexion the hyoid muscles come into play, innervated by the branches from the descendens and communicans hypoglossi, C^3, C^4, anterior branches.

The movements of *extension* are caused by the complexus, trachelo-mastoid splenius, obliquus inferior and recti capitis postici and somewhat by the trapezius. These are innervated practically entirely by the posterior branches of the cervical nerves—from C^1 to C^3.

The *lateral* movements of the head are caused by the sterno-mastoid, splenius, scaleni, trapezius, complexus, obliquus superior, rectus capitis lateralis, supplied by the spinal accessory, posterior branches of the cervicals C^1 to C^3, except the recti laterales, supplied by the anterior branches C^1.

Extension and lateral movements of the head are mainly done by the spinal accessory and upper three cervical posterior branches. The sterno-mastoid gets some branches from the anterior cervical division C^2, as do the recti capitis laterales.

Rotation of the head is caused by the sterno-mastoid, splenius trachleo-mastoid, complexus, obliquus superior and inferior, recti capitis postici and levator anguli scapuli. These are supplied by the spinal accessory and posterior branches of the cervicals C^1 to C^1, except the sterno-mastoid and recti capitis laterales, which is supplied by the C^1 and C^1 anterior branches.

Hence, after divsion of the posterior branches of the cervical nerves C^1 to C^4 there still remain innervations of the recti capitis laterales and levator anguli scapuli, sterno-mastoid and trapezius. When the spinal accessory is cut these other innervations remain and the head may yet be extended, bent laterally and rotated.

The muscles which rotate the head to the same side are the recti capitis lateralis (slightly), levator anglui scapuli, trachleo-mastoid (posterior C^3), complexus (posterior C^1, C^2, C^3) obliquus inferior (posterior C^2). See diagram, Fig. 56.

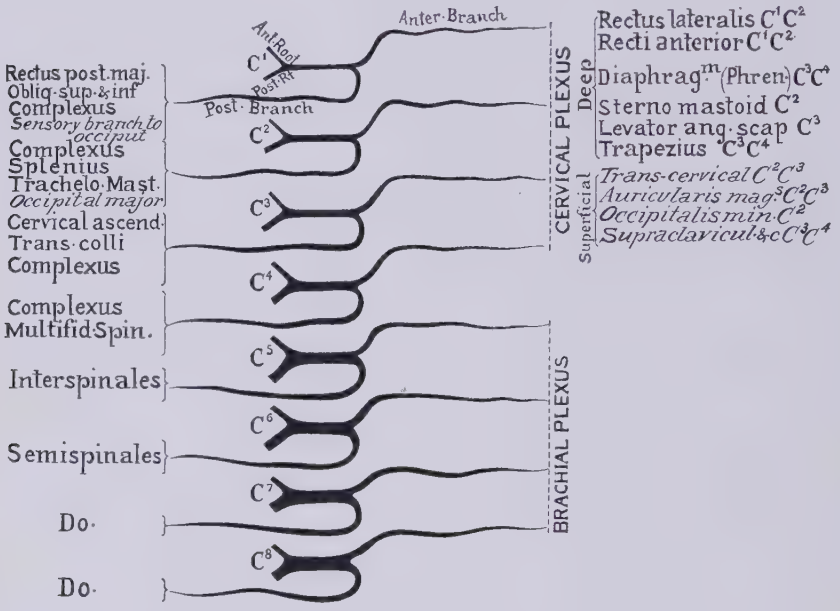


FIG. 56.—Diagrammatic representation of the anterior and posterior divisions of the cervical nerves.

Those rotating to opposite side are the sterno-mastoid, obliquus superior (posterior C^1).

The diseases of the upper cervical group are spasms, paralyses and neuralgias.

Spasmodic Diseases.—*Torticollis* may be limited to and is usually associated with the cervical nerves, as has been shown, though it is usually primarily a spinal accessory neurosis.

Tonic spasm causing a rigid neck is a frequent symptom of meningitis, and forms part of epileptic and other convulsions.

In oscillatory and rotatory spasms of the head the cervical nerves are involved.

Nodding Spasm, Oscillating Spasm.—This is a disorder occurring chiefly in children and characterized by rhythmical nodding or oscillating movements of the head. It occurs, however, in adults.

Etiology.—The disease occurs in young children who are anæmic and badly nourished. Dentition, digestive disorders, eye defects, are causes. Sometimes it is only a kind of habit spasm, and occasionally this habit continues during life. It may be associated with or be a precursor of torticollis. It occurs in hysteria, and sometimes in elderly people forming part of a family or senile tremor.

Symptoms.—The condition in young children is usually a constant slow, mild, rhythmical, oscillating spasm. It is usually "outgrown." In adults it may assume the chronicity of a torticollis or it may be plainly part of a general senile or family tremor.

The *diagnosis* is easily made by the symptoms. The *prognosis* and *treatment* depend upon the etiology. I have found bromide of potassium and hyoscine useful in elderly cases, and syrup of iodide of iron in children.

The *phrenic* nerve arises from the third, fourth and fifth cervical, oftenest from the fourth and fifth (37 per cent.). Next from the fourth alone (25 per cent.) and then from the third, fourth and fifth (23 per cent.) and from the third and fourth (15 per cent.) (Schroeder-Green).

The diaphragm is supplied by a few filaments of the intercostal nerves, but these cannot take the place of the phrenic and their function is slight (Cavalie).

In some forms of *stuttering* the phrenic nerve is involved in a clonic or tonic spasm.

Hiccough is a clonic spasmodic disorder of the phrenic nerve. It is usually due in its casual form to gastric disturbance, with flatulent distention of the stomach.

When more chronic, it is caused by hysteria, neuritis, diaphragmatic pleurisy, or some pressure upon the nerve in its course. It is sometimes a prælethal symptom. I have seen cases in which it was probably a pure spasmodic neurosis, a form of tic.

Ordinarily, hiccough can be stopped by simple carminatives like spirits of chloroform or lavender or a hot milk punch. In obstinate cases in which no known cause can be found, pilocarpin, hyoscine and bromides are useful. A most effective measure is to lay the patient supine over a thick bolster so that the head hangs back and the thorax arches up. An injection of morphine and atropine promptly stops some cases.

Paralysis and Atrophy.—The cervical muscles are paralyzed in anterior poliomyelitis, progressive muscular atrophy, in pachymeningitis

hypertrophica, and occasionally in vertebral and peripheral disease or injury and in spinal-cord tumors. Some deformities and weakness in head movements result, but the most serious consequence is involvement of respiration through palsy of the phrenic.

Paralysis of the Phrenic Nerve—Etiology.—Such paralysis may be due to disease or injury of the cervical cord and also to peripheral disease, to which the nerve is somewhat liable owing to its long course through the anterior mediastinum.

Pleurisy, peritonitis, mediastinal tumors, and multiple neuritis are among the special causes of phrenic paralysis. Spinal-cord diseases such as tabes, acute ascending paralysis, and surgical injuries are, however, the commonest etiological factors.

Symptoms.—In diaphragmatic paralysis, if bilateral, as is usually the case, the epigastrium and hypochondrium are drawn in in inspiration instead of rising. On slight exertion there are dyspnœa and increase of respiration. The cough is very weak.

Diagnosis.—If no other muscles than the diaphragm are involved, the cause is probably in the trunk of the nerve. Inflammatory disease of the diaphragm may cause a paralysis which is recognized by its painful character and the febrile reaction.

Treatment.—This is to be guided by the cause. It need only be said that there is a motor point in the neck where by careful electrization one can get a contraction of the diaphragm. In paralysis of the phrenic this fact should be borne in mind.

GROUP II. THE LOWER CERVICAL NERVES AND BRACHIAL PLEXUS

Anatomy and Physiology.—The anterior branches of the lower four cervical nerves and first dorsal nerve unite to form the brachial plexus. This gives off *short nerves* to the shoulder and trunk and *long nerves* to the arm.

The mode of formation of the brachial plexus is shown in the diagram (Fig. 57). It is in accordance with the description by Cunningham. The *short* or upper branches supply the shoulder and intercostal muscles. The *long* or lower branches supply the arm and hand. The neurologist needs to know: (1) the muscular distribution of each nerve and the function of the muscle; (2) the cutaneous sensory distribution; and (3) the level of origin of the nerves.

The Plate II, Jakob, Fig. 57, and the table, p. 132, give these points, and will be found useful for study and reference. They are based upon the investigations of Ferrier and Yeo, Thorburn, and also on Abbe's and my own experiments.

The Arrangement of the Brachial Plexus.—It is made up of three nerve trunks which in turn make up three cords, these cords giving off various branches, thus:

1. Trunk from sixth and seventh cervical roots	forms outer cord, which gives off	Ext. ant. thoracic. Musculo-cutaneous. Outer head median.
2. Trunk from eighth cervical and first dorsal roots	forms inner cord, which gives off	Inner head median. Ulnar. Int. cutan. Int. ant. thorac.
3. Trunk from fifth, sixth, seventh, and eighth cervical and first dorsal	forms posterior cord, which gives off	Intercost.-hum. Subscapular. Circumflex. Musculo-spiral.

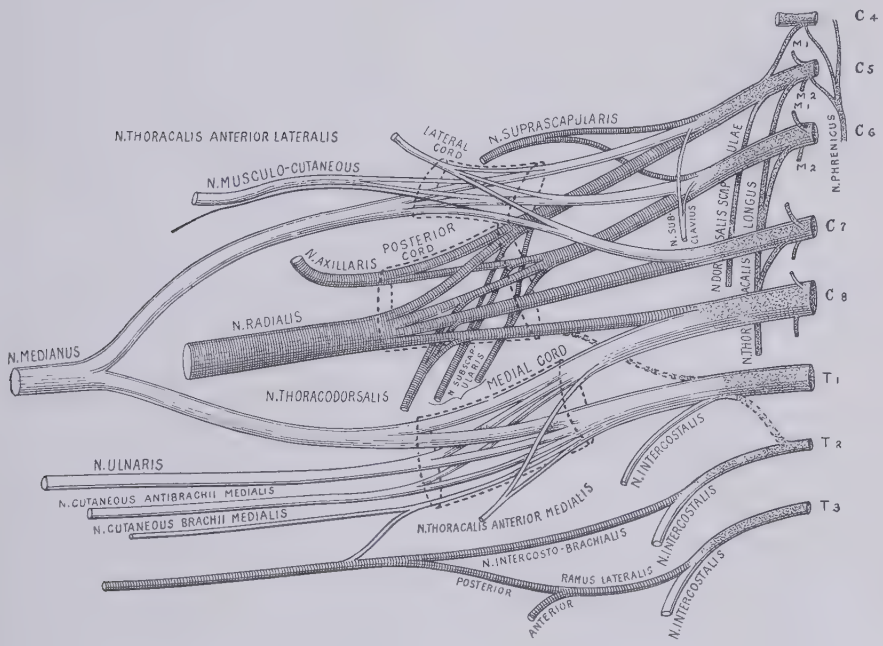


FIG. 57.—The brachial plexus. (*Cunningham.*)

The following table shows the origin, muscular distribution, and effect of paralysis on the motor but not on the sensory nerves. This latter is indicated in Figs. 27 and 28.

Nerves and Roots of Origin	Muscular Distribution	Function as Shown by Effect of Paralysis
Posterior thoracic. Fifth and sixth cervical.	Serratus magnus.	Posterior edge of scapula is rotated out when arm is raised and carried forward. Weakening of elevation of shoulder and of inspiration.
Circumflex. Fifth and sixth cervical.	Deltoid. Teres minor.	Loss of power to raise arm.
Suprascapular.	Supraspinatus.	Weakened power to raise arm; head of humerus tends to fall.
	and	Loss of abductors; motion forward, and inward rotation of humerus.
	Infraspinatus and teres minor.	Loss of rotation of humerus outward.
Subscapular, short. Fifth and sixth cervical.	Subscapulares. Teres major.	Weakens inward rotation of arm. Weakens power of elevating shoulder.
Subscapular, long. Fifth, sixth, seventh and eighth cervical.	Latiss. dorsi.	Weakens power to depress shoulder and to pull arm backward and to side.
Anterior thoracic.	Pectoralis major.	Loss of power to pull arm down and forward and to shrug the shoulder.
Musculo-cutaneous. Fourth and fifth cervical.	Biceps and brachialis anticus.	Loss of flexion of forearm.
Musculo-spiral. Fourth, fifth, sixth, seventh and eighth cervical.	Triceps. Supinatores.	Loss of extension of forearm. Loss of supination.
	Extensor carp. rad. Extensor carp. uln.	Extension of wrist lost except when fingers are flexed; lateral deviation.
	Extensor comm. digit.	Impaired extension of first phalanges and wrist.
	Extensor p. i. pollic. Extensor s. i. pollic. Extensor oss. met. pollic.	Impairment of extension of thumb.
Median. Fifth, sixth, seventh, and eighth cervical.	Pronatores. Flexor carp. rad. Flexor sublim. dig. Flexor profund. dig. radial half. Two lumbricales.	Loss of pronation. Weakened flexion of wrist. Weakened flexion of second and third phalanges of first and second fingers.
	Abductor pollic. Flexor pollic. Flexor carp. ulnar.	Loss of abduction and flexion of thumb. Weakened flexion of wrist; radial deviation.
Ulnar. Eighth cervical, first dorsal.	Flexor profund. dig. ulnar half.	Weakened flexion of second and third phalanges of third and fourth fingers.
	Interossei. Two lumbricales. Flexor minor digit. Adductor pollicis. Inner half of flexor brev. pollicis.	Loss of flexion of first phalanges and of extension of second and third.

Diseases of the Lower Cervical Nerve Group and the Brachial Plexus.—The nerves of this group are subject to the pathological disturbances common to all nerves. I shall describe them from the clinical side, which gives the following disorders:

Paralyses	Combined arm palsies.
	Upper-arm type or Erb's paralysis.
	Lower-arm type. Klumpke's paralysis.
	Paralyses of individual nerves.
Spasmodic Disorders	Occupation neuroses
Sensory Disorders	Brachial neuralgia.
	Digital neuralgia.
	Numb hands (acroparæsthesia).
Secretory, Trophic, and Vasomotor Disorders	Neurotic œdema.
	Symmetrical gangrene.

Spasmodic Disorders of the Arm.—The arms and hands are especially subject to tremors, choreic and other spasmodic movements. The only spasmodic disorders, however, which may be said to be especially located there are writer's cramp and allied occupation neuroses. These are described elsewhere.

Brachial Paralyses, Arm Palsies.—These occur as combined or total-arm palsies, upper-arm and lower-arm types, and paralyses of single nerves.

Combined paralysis of the brachial nerves is a condition in which all or nearly all the portions of the plexus and its branches are involved. Total-arm palsies make up about 6 per cent. of all peripheral paralyses, and are about one-fifth as frequent as single-nerve paralyses.

Etiology.—They occur oftenest in men, but are not rare in infants, being then due to injuries during parturition. After infancy they are most frequent in early and middle life.

The exciting causes are obstetrical injuries; wounds, cuts, fractures; deep-seated inflammations and tumors; shoulder dislocations; primary neuritis; crutch and other forms of mechanical compression; hysteria; in rare cases, spinal-cord and brain disease.

The *symptoms* vary with the severity and extent of the lesion.

With regard to severity, there are three degrees. In the first there is simply a transient palsy, due to lying too long on the arm. The arm feels heavy, numb and "asleep." In a few minutes or hours this palsy disappears. In the second degree the nerves are so much compressed as to be mechanically injured. If the patient has been drinking hard, even moderate pressure may set up an inflammatory or destructive process that leads to quite a serious palsy. In the third degree the nerves are actually cut or torn across, or so compressed as to lose their anatomical integrity.

The resulting symptoms are those common to all nerve injuries, viz., paralysis, wasting, changes in electrical reaction of the muscles. Pain, tenderness, anæsthesia, trophic, secretory and vasomotor disturbances are also present in varying degree.

The distribution of the paralysis, atrophy, and sensory disturbance depends, of course, upon the arm nerves chiefly involved. The cuts and table will enable one to see in any case where the trouble is localized.

It is important to determine whether one is dealing with a total-arm palsy, an upper-arm type (Erb's palsy), or a lower-arm type.

In *Erb's palsy* there is involvement of the deltoid, biceps, brachialis anticus and supinator longus, with at times paralysis of the supinator brevis, infraspinatus and even of all the muscles supplied by the median nerve. The lesion is either in the cord formed by the fifth and sixth

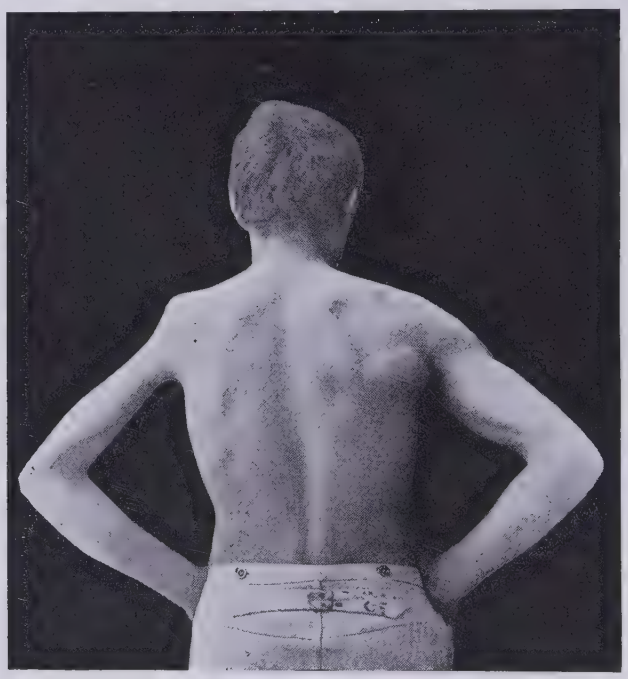


Fig. 58.—Upper-arm palsy of left side.

cervical nerves or a little lower in the brachial plexus, where the fibres supplying the musculo-spiral, circumflex and musculo-cutaneous lie close together. At all events, the lesion involves the central parts and upper cords of the plexus. The arm hangs by the side and the forearm cannot be flexed (Fig. 58). There are many variants of the upper-arm-shoulder paralysis due to surgical injuries, tumors, passive hanging by the hands by gymnasts, etc.

The upper-arm type is especially frequent in infants and constitutes one of the obstetrical paralyses. The trouble is caused through compression and tearing of the upper cords of the plexus in difficult labor. Among thirty cases collected by Dr. A. S. Leverty, twenty-eight were

difficult head presentations. Sometimes there is a hemorrhage in the cord itself.

In the *lower-arm type* (Klumpke's paralysis) the triceps, the flexors of the wrist, the pronators, the flexors and extensors of the fingers and the hand muscles are involved. The arm can be raised and the forearm flexed and supinated, but the hand is useless and the extension of the forearm is impossible. The lesion here involves chiefly the nerves from the seventh and eighth cervical and first dorsal roots (Fig. 59).

If the lesion is in the nerve there will be atrophy, changes in electrical reaction, sensory disturbances, and often, if there is neuritis, pain over the nerves. The reflexes will be lessened or absent. If the lesion is in the spinal cord, symptoms in other parts of the body will be present, or,

Fig. 59.—Lower-arm palsy and wrist-drop.

if not, there will be no sensory disturbance, as in an arm palsy from anterior poliomyelitis. In rare cases arm palsies may be caused by spinal tumors or a local meningitis, in which case the oculo-pupillary centre of the cervical cord and the sympathetic may be involved.

There are three common symptoms, however, of which it is often very important to analyze the cause. These are the loss of power for elevation of the arm and for flexion and extension of the forearm.

Flexion of the forearm is performed by the biceps and brachialis anticus, and is helped by the supinator longus. These muscles are supplied by the musculo-cutaneous nerve, except the supinator, which is supplied by the musculo-spiral. Hence when a person cannot flex the forearm, the musculo-cutaneous is chiefly affected.

Extension of the arm is done by the triceps, which is supplied by the musculo-spiral.

Elevation of the Arm Outward.—Inability to raise the arm is the common and striking symptom in combined brachial palsies. The arm is raised by a number of muscles. The deltoid acts first and most, but it can raise the arm only to a right angle. It is supplied by the circumflex nerve from the posterior cord of the plexus. After the arm is raised to a right angle, it is further elevated by rotating the scapula, and this is done chiefly by the middle part of the trapezius (lower cervical and upper dorsal nerves) and by the serratus magnus, supplied by the posterior thoracic nerve. A number of other muscles combine to strengthen elevation of the shoulder, but this action can be abolished only by paralysis of the deltoid or trapezius and serratus magnus.

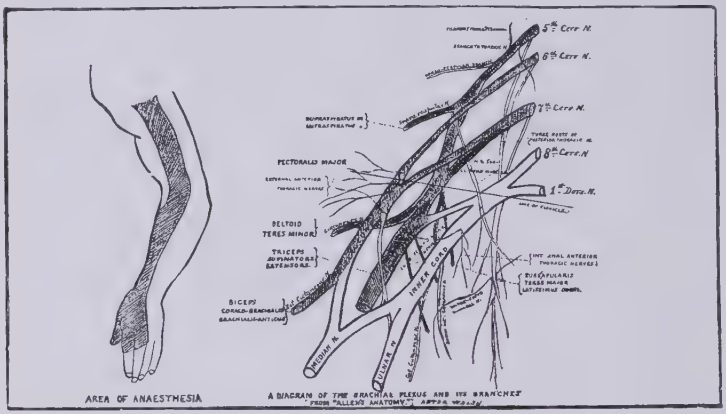


FIG. 60.—Illustrating the formation of the brachial plexus; also the involvement of the plexus in degenerative neuritis. (*Leszynsky.*)

The *diagnosis* of these cases involves, first, consideration of the seat of the lesion and the special nerves involved; next, that of the nature of the lesion. A recognition of the seat of the lesion and of the special nerves involved depends entirely upon the study of the distribution of the palsy and of the atrophy and sensory disturbance.

The *prognosis* in arm palsies as a whole is usually good except for the total-arm palsies. Nearly all these cases get well, the duration of the incapacity being from two or three months to a year. Even in the severest cases recovery is possible after one or two years. If, however, the nerves are torn across, as in birth palsies, and the ends widely separated, recovery is impossible unless an operation is promptly done.

The *treatment* consists in electrical applications, educational movements, mechanical support, with potassium iodide internally and absti-

nence from alcohol. Local injections of nitrate of strychnine are useful, and massage should be used if it can be applied carefully.

In brachial palsies due to severe injuries, dislocations, fractures, etc., in which there is evidence, from the extreme atrophy and absence of electrical reaction, that the nerve is entirely cut across and that the ends are not in apposition, a surgical operation is stringently needed. The nerves should be exposed and the ends brought as near together as possible. When this cannot be done, the nerves can be split and the ends sutured or anastomoses made, or the two ends are sewed together as

Fig. 61.—Double paralysis of circumflex nerve.

near as possible and placed in a tube. If the separation is over an inch, not much can be expected. In all these cases, however, it must be remembered that the two ends do not unite, but the central end grows down in the tract of the old degenerated peripheral stem.

Paralysis of Single Nerves—Paralysis of the Posterior Thoracic Nerve—*Etiology.*—This rare trouble usually occurs in male adults and is due to injury or sudden strains. Its paralysis may be part of a progressive muscular atrophy. The nerve goes to the serratus magnus.

Symptoms.—When paralyzed, there is difficulty in raising the arm above the horizontal position and the movements of the shoulder are

weakened. When the arm hangs by the side the lower angle of the scapula is drawn a little nearer the vertebral column and protrudes slightly. When the arm is held out horizontally the inner edge of the scapula protrudes and is drawn toward the middle line. When the raised arm is brought forward there is a deep groove formed between the inner border of the scapula and the thoracic wall (Fig. 65).

The disease often runs a long course and is accompanied by pain.

Paralysis of the Circumflex Nerve.—The nerve goes to the deltoid, teres minor, third head of the triceps and shoulder-joint. It gives sensation to the skin of the shoulder. It is very often paralyzed. The commonest causes are a fall or injury, dislocation and rheumatic inflammation of the joint. The arm cannot be elevated or rotated outward (Fig. 61). There are atrophy, anæsthesia and sometimes pain.

Paralysis of the Suprascapular Nerve.—The nerve goes to the spinati muscles, teres minor and shoulder-joint. Disease of this nerve alone is rare.

The supraspinatus rotates the shoulder in, the infraspinatus and teres minor rotate it out. When paralyzed, there is an impairment of rotation and some impairment of elevation of the shoulder.

Paralysis of the Musculo-spiral Nerve (*Wrist-drop, Lead Palsy, Compression Paralysis*).—The distribution of this nerve is given in the table and cut. Its function is to extend and supinate the forearm, to extend the wrist and fingers, and slightly to adduct and abduct the fingers. It extends directly only the last or ungual phalanges, the first and second phalanges being extended by the ulnar nerve.

Etiology.—The musculo-spiral, owing to its course, is the most frequently affected by paralysis of all the arm nerves. Pressure on the nerve during sleep—especially when the patient is intoxicated, crutch pressure, fractures, wounds, tumors, lead poisoning, arsenical, alcoholic and other forms of multiple neuritis are the causes of its disordered function.

Symptoms.—The symptoms of this paralysis are "wrist-drop," due to an inability to extend the wrist or fingers. The first and second phalanges can be extended somewhat by the interossei and lumbricales, but the last phalanges cannot be extended at all. The first finger is least affected. The fingers can be only slightly abducted, supination is generally lessened or lost; if the lesion is high up, the triceps is involved and the power of extending the forearm weakened. There may be atrophy of the muscles and degeneration reaction. A swelling over the tendons of the wrist-joint may occur. Some numbness and tingling exist, and occasionally there is anæsthesia in the distribution of the radial nerve on the hand. The disease lasts but a few weeks if due to com-

pression; for months if due to neuritis, lead poisoning or severe injury of the nerve. Eventually recovery takes place, as a rule.

When the disease is due to *lead poisoning* there are some peculiarities in its course. Thus the supinator longus usually escapes; the palsy begins gradually and usually involves both arms; it may extend to the upper arm. Partial degeneration reactions are present. There is rarely any anæsthesia and but little pain. Often there is a lead line on the gum and a history of constipation and colic.

In *alcoholic* and other forms of multiple neuritis there are pain and paræsthesia, both arms are involved, and the flexors and other fore-

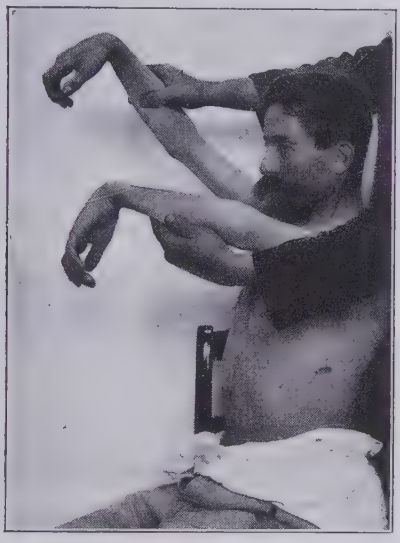


Fig. 62.—Wrist-drop from lead palsy.

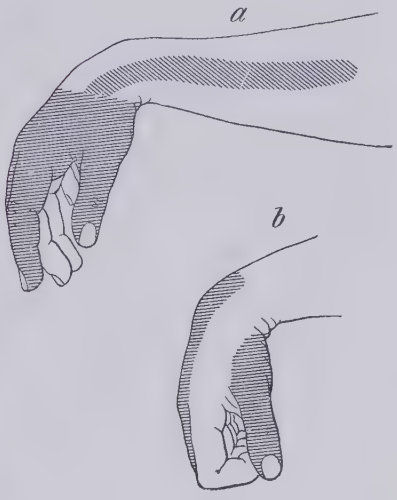


Fig. 63.—(*a*) Paralysis of radialis and median nerves with anasthesia (*b*) paralysis of radialis. (*Leverty.*)

arm muscles are somewhat implicated. There are marked sensory disturbances. The legs are also affected.

In *compression palsy* the supinators and often the triceps are involved.

When the lesion of the nerve is high up, as in *crutch paralysis*, there is but little anæsthesia, and that is found on the anterior surface of the forearm, in the distribution of the external and internal cutaneous nerves. Lesion of the nerve lower down may give rise to some anæsthesia along the radial border of the forearm and back of hand, but the anæsthetic area varies a great deal.

The *diagnosis* of the paralysis is easily made. The most important point is to find out the cause. The different characteristics of lead palsy, neuritic palsy and compression palsy have been indicated in the

description of the symptoms. One must be sure to exclude also progressive muscular atrophy.

The *treatment* consists of mechanical measures, such as electricity, massage, the application of rubber muscles and in bad cases the fixation of the forearm and hand in hyperextension by means of a splint and plaster-of-Paris bandage (Gibney). Internally in the early stage it is best to administer iodide of potassium and sulphate of magnesium (in lead palsy), the salicylates in neuritis; later, hypodermic injections of strychnine may be given. Static sparks, galvanism and other forms of electricity unquestionably do good in some cases.

Paralysis of the median nerve is rare as an isolated trouble, and is almost always due to injury or neighboring disease.

When paralysis occurs the grip is weakened. Flexion and abduction of the thumb and flexion of the first and second fingers are impaired. Atrophy of the thenar eminence may occur. The anæsthetic area varies, but is shown in the accompanying cut (Fig. 63).

Paralysis of the Ulnar Nerve—Etiology.—The ulnar nerve is rather commonly affected by paralysis, the occurrence ranking next in fre-

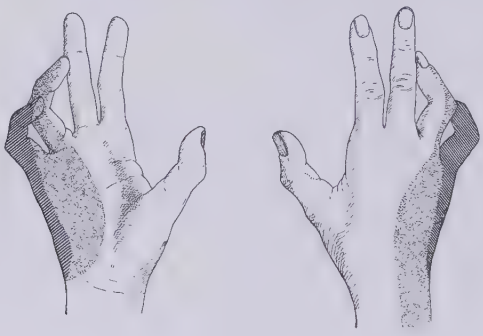


Fig. 64.—Paralysis of ulnar.

quency to musculo-spiral palsy. It is rarely affected in lead poisoning, but is usually early involved in progressive muscular atrophy. It is sometimes attacked by a primary degenerative neuritis. Injuries are the common cause.

The *symptoms* are shown by the table (p. 130). The hand cannot be closed tightly, the little and ring fingers being especially weak. The first phalanges are drawn back and the second and third phalanges flexed; when the interossei and lumbricales atrophy, the result is the "griffin claw" or *main en griffe*. The fingers cannot be adducted or abducted except feebly.

There is anæsthesia over the area of distribution of the ulnar (Fig. 64); there may be pain and tenderness.

Migrating neuritis is a serious but rare malady which deserves some mention. It occurs as a sequel to some wound of or operation upon a nerve. The local neuritis extends usually up the arm (ascending neuritis). It is accompanied by intense pain, anæsthesia, paralysis and atrophy. The disease is very chronic and intractable. It has been relieved in some cases by resecting the posterior spinal roots.

Morvan's Disease, Analgesic Paralysis with Whitlow.—(Neuritic type of syringo-myelia.)

This is a very rare disease, characterized by a slowly progressive paralysis and atrophy of the hands and forearms, with analgesia and painless whitlows. It is probably always either a form of syringo-myelia or of leprosy.

FIG. 65.—Paraiysis of right posterior and thoracic nerve and serratus magnus muscle.

GROUP III. THE THORACIC OR DORSAL NERVES

Anatomy and Physiology.—The dorsal nerves are twelve in number. The first is the largest and belongs functionally to the arm nerves. The dorsal nerves carry motor and sensory fibres to the voluntary muscles, skin and other tissues of the trunk wall. They carry some splanchnic fibres to the lungs and abdominal viscera. They divide into anterior and posterior branches. The anterior form *intercostal nerves*, of which the first six are distributed to the wall of the thorax and the last six to the wall of the abdomen. All these nerves give off lateral and anterior branches. The posterior branches of the dorsal nerves are small and supply the muscles and skin of the back.

The upper six dorsal nerves, including both branches, are mainly inspiratory in function. They also extend and rotate the dorsal and cervical vertebræ. The lower dorsal are expiratory nerves; they also assist in compressing the abdominal viscera and in flexing, extending and rotating the spine.

Motor Neuroses.—The thoracic motor nerves are mainly involved in respiratory cramps and paralyses; sneezing, coughing, laughing and crying are symptoms in which they play a large part. In complete respiratory paralysis also these nerves are affected. But there are few motor neuroses that are limited to the thoracic nerves. The neuroses of these parts are mainly sensory and will be described later.

GROUP IV. THE LUMBAR NERVES

Anatomy and Physiology.—The lumbar nerves are five in number. The posterior branches supply the erector spinæ, interossei, multifidus spinæ and interspinales, and

also the skin of the back. The anterior branches of the upper four unite to form the *lumbar plexus*. The fifth or lumbo-sacral nerve sends most of its fibres to the sacral plexus. The branches of the lumbar plexus are: (1) the ilio-inguinal, (2) ilio-hypogastric (from first lumbar), (3) genito-crural, and (4) external cutaneous (mainly from the second), (5) obturator (from third and fourth lumbar), (6) the anterior crural (from second, third and fourth—Plate II).

The first four branches of the plexus are comparatively short and supply sensation

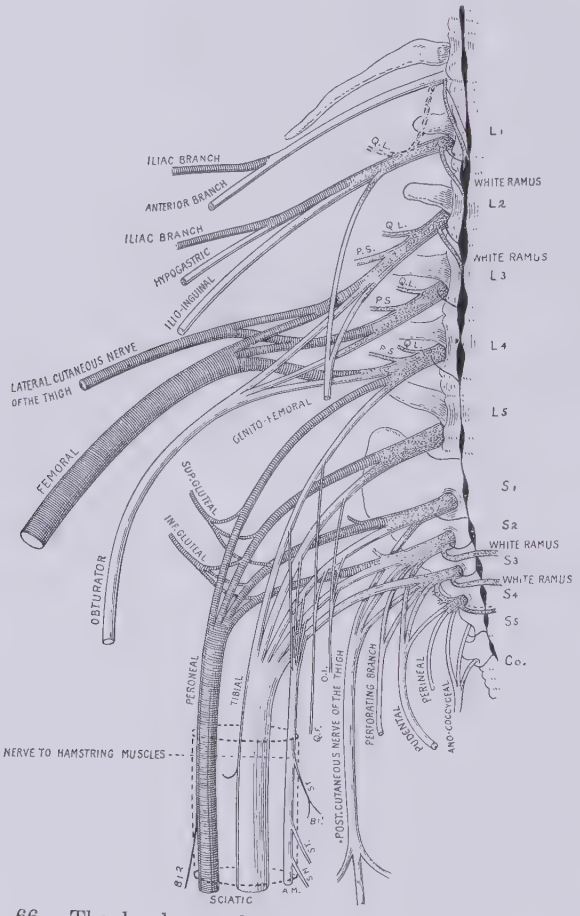


FIG. 66.—The lumbar and sacral plexuses. (*Cunningham.*)

to the abdominal wall and external genitals. The last two are longer and supply the hip- and knee-joints, the muscles of the anterior inner and outer part of the thigh, the skin over this region and the inner side of the leg and dorsum of the foot (Figs. 27 and 28).

The diseases of the lumbar nerves and plexus so far as they form independent disorders are mainly neuralgias. In making a diagnosis of lumbar-nerve disease, one should remember that of the six branches of the plexus the upper four are mainly sensory, the lower two mixed nerves.

Paralyses and spasmodic troubles *of the lumbar nerves* are not rare, but are usually symptomatic of some extrinsic and often serious disease.

Etiology.—Hence it is well to catalogue here those affections which may produce lumbar palsies or spasms. They are pelvic tumors or injuries, impacted fæces, caries of the spine, psoas abscess, obturator hernia, hip disease and pressure of the fœtal head.

Symptoms.—When the upper two lumbar nerves are involved, only sensory symptoms in the distribution of their branches occur. If the next two are also involved, there may be trouble in extending the leg and flexing the hip on the trunk. The patient cannot raise himself to a sitting posture. If there are irritation and spasm, the thigh is drawn up and adducted.

In *paralysis of the obturator nerve* there are loss of power to adduct the thigh and cross the leg and weakness of outward rotation of the thigh. Anæsthesia over the inner side of the thigh may be present.

In *paralysis of the anterior crural nerve* there are weakness of the muscles of the anterior region of the thigh, loss of power of extending the leg, and anæsthesia or pain over the crural area.

Paralysis of the posterior branches of the lumbar nerves causes weakness or paralysis of the erectors of the spine. The lumbar curve is very greatly exaggerated, the shoulders being thrown far back and the belly protruding. This condition occurs in progressive muscular dystrophy, particularly in the pseudo-hypertrophic form.

GROUP V. THE SACRAL NERVES

Anatomy and Physiology.—The sacral nerves are five in number. The first four divide into anterior and posterior branches. The fifth has no anterior branch. The posterior branches escape through the posterior sacral foramina and supply the multifidus spinæ and the skin over the sacrum and coccyx.

The first three anterior branches, with the lumbo-sacral nerve and a branch from the fourth sacral, unite to form the sacral plexus. This lies upon the pyriformis muscle in the pelvis, and escapes at the lower part of the sacro-sciatic foramen. The great mass of the fibres go to make up the sciatic nerve.

The roots of origin of the sacral and coccygeal nerves form the cauda equina.

The branches of the sacral plexus are the superior gluteal, muscular, small sciatic, inferior gluteal, pudic, great sciatic, perforating, cutaneous and articular. These are distributed to the muscles, skin and joints of the buttocks, thighs, legs and feet. The sensory distribution is shown in Figs. 27 and 28.

The sacral nerves are the main agents in station and locomotion. They control the legs entirely, also the posterior muscles of the thigh and buttocks; they give sensation to these parts. They carry also fibres that regulate the sexual function, bladder and rectum. From the sacral portion of the cord there is an outflow of nerves to the autonomic system, thence to the pelvic organs.

The diseases of the sacral nerves may be classified in a similar way to those of the brachial plexus.

Spasmodic Disorders of the Sacral Nerves.—Tremor, rigidity, clonic and tonic spasms, myoclonus, athetoid movements all affect the lower extremities, but they are almost invariably part of some general or central disorder, such as chorea, paralysis agitans, hysteria, etc. Under the head of occupation neuroses there occur certain rare spasmodic troubles special to the legs. Saltatory spasm involves the legs alone. These disorders are, however, general neuroses.

Peripheral Leg Palsies.—Paralyses of the lower limbs from disease of the nerves may be either combined or single, just as is the case with arm palsies.

A combined sacral palsy is one in which all or nearly all of the branches of the sacral plexus are involved.

Etiology.—Such palsies are due to much the same causes as those affecting the lumbar nerves, viz., injury, dislocation, hip disease, tumors and neuritis. Hysteria may cause a functional sacral palsy.

Symptoms.—The symptoms are indicated by a study of the distribution of the nerves, varying, however, in degree. The foot cannot be moved; the leg can be slightly extended by the anterior crural, but not flexed; the thigh cannot be extended freely or rotated perfectly. There is anæsthesia over the distribution of the sacral nerves; pain may be present; wasting and vasomotor and secretory disturbances occur unless the paralysis is functional.

The course depends on the severity of the lesion. If the nerve is totally cut or torn across, it may require one or two years for perfect recovery which, however, occurs if the severed ends are properly approximated.

The *diagnosis* of a sacral palsy is based on the history and on the distribution of the anæsthesia and of the muscular paralysis. The sacral nerves do everything for the lower limb except extend the leg, flex and adduct the thigh, and to some extent rotate it. They supply sensation equally extensively.

The distinction from spinal-cord disease is chiefly based on the unilateral symptoms, the absence of disorder of the sphincters and the combination of paralysis, wasting and sensory troubles in the course of the sacral nerves.

Single-nerve Sacral Palsies.—The symptoms of paralysis of single nerves are indicated by their function. The nerves rarely affected are the superior gluteal, muscular and small sciatic. The nerve oftenest affected is the *great sciatic*, and especially its *anterior tibial branch*. In the latter case a condition called "drop-foot" is produced.

In the *pathology* and *treatment* of sacral palsies there is nothing especial that can be said.

CHAPTER X

SENSORY NEUROSES OF THE CEREBROSPINAL NERVES

As the most common disorders of the sensory nerves are neuralgias and paræsthesias, I shall introduce the subject with a general description of these symptoms.

PARÆSTHESIA

LOCAL PARÆSTHESIA ACRO-PARÆSTHESIA, WAKING NUMBNESS

The condition known as paræsthesia is one which should be more familiar to physicians and be more commonly recognized and understood. *Paræsthesia* is the name given to a number of subjective sensations, such as prickling, numbness, creeping sensations, tickling and burning. It includes, in fact, nearly all the subjective sensations of the skin, except those of pain. It is a condition which is, therefore, extremely common, and in its mildest and most trivial character is much more often experienced than pain. When these sensations fix themselves in a certain locality, following perhaps the tract of the nerve or fastening themselves upon the hand or foot, they take on a certain clinical type, and deserve to have the name of a disease to just the same extent that a neuralgia does. Paræsthesia, in almost all cases, implies simply a lower grade of irritation of the nerve-fibres that occurs in neuralgia, and is a kind of ghostly simulacrum of that disease. It very often precedes or accompanies attacks of pain. There is sometimes a tingling of the teeth or burning in the face which has a shadowy likeness to a toothache or trigeminal neuralgia. In the same way, one finds paræsthesias affecting the head, causing sensations of pressure and constriction, of burning, and general undefinable discomfort, which are entirely comparable to headaches.

In conditions of neurasthenia, paræsthesias of the head are more common even than the headaches. Paræsthesia sometimes follows the course of a nerve, as when one feels numbness of the hand if the ulnar is pressed upon at the elbow, or numbness in the foot where the sciatic is pressed upon, as when the legs are crossed.

There is also paræsthesia affecting one of the intercostal nerves or one of the crural nerves. On the other hand, paræsthesia may affect all four extremities, so that they feel entirely benumbed or prickling.

Paræsthesia is usually peripheral and neuritic, but it may be cerebral and due to cord or brain disease.

Etiology.—Paræsthesia, whether local or diffuse, occurs rather more often in women than in men, and rather more often in the mature and middle-aged than in young people. It is most frequent in women of middle age, especially in those who are accustomed to hard work with the hands in washing and the exposure incidental to this. It also occurs in those who are obliged to walk a great deal and to be upon their feet, and it especially affects tailors, seamstresses, bookbinders and those who have to use their hands constantly in some skilled mechanical work. It sometimes occurs in old age, being accompanied by evidence of gout or by glycosuria. It is associated with rheumatism and with alcoholism. It follows infections like typhoid fever and the grippe, and means in these, as in many other cases, a low grade of neuritis.

Paræsthesias which at first are due to some direct or reflex nerve irritation may become a habit and form a part of a psychosis. Paræsthesias are very common in melancholia and hypochondria.

Cerebral paræsthesias occur in tabes, combined sclerosis and in thalamic lesions.

Paræsthesia affects single cerebrospinal nerves just as neuralgia does, or it may be more generally distributed. In the latter case it affects often the feet and hands, and it is called *acro-paræsthesia.*

We meet then with:

1. Cephalic paræsthesias, comparable to diffuse headaches.
2. Local paræsthesias, comparblae to local neuralgias.
3. Acro-paræsthesia, involving the feet or hands or both diffusely.
4. Psychic and cerebral paræsthesias.

The cephalic paræsthesias are usually symptoms of neurasthenic or depression states.

Acro-paræsthesia occurs in multiple neuritis, in glycosuria, and pernicious anæmia.

Local forms affect the ulnar, trigeminal, cervical, sciatic and lumbar nerves oftenest.

Under the head of paræsthesic neurosis, the affection known as *waking numbness* or *night palsy* may be described. This is a disorder characterized by a temporary paralysis of an extremity, with numbness, noticed on first waking or after lying down for a time. It is a rare disease and little is known of its cause. It occurs in adults and usually in the neuropathic. Sometimes evidence of arthritism, arterial sclerosis or poor innervation of the vessels is present.

The symptoms are much like those caused by temporary compression of a nerve when the leg or arm goes to sleep. The paralysis is

temporary and there is no anæsthesia. It is often a very obstinate condition but leads to no serious result.

In some cases there is in paræsthesia undoubtedly a low grade of neuritis, and in other cases there is a congestion or slight degree of degeneration of the nerve. Underlying many of the paræsthesias of middle and late life is an arterial sclerosis or defects of metabolism with nerve degeneration. Many of the cold and heat sensations of this period have this vascular origin, to which is added an undue mental irritability and sensitiveness.

NEURALGIA

Neuralgia is a condition characterized by pain in the course of a nerve or of nerves. It is not a distinct disease, but only a symptom,

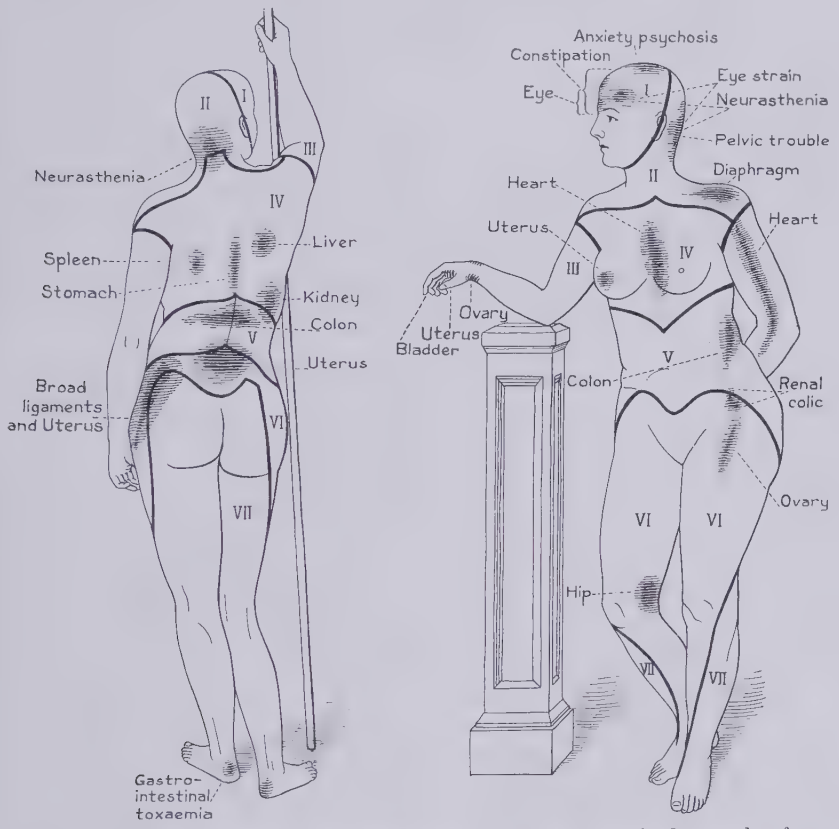


Fig. 67.—Diagrams showing the distribution of the cerebrospinal strands of nerves and the location of transferred pains and neuralgia.

and the tendency is to do away with the term except to indicate a nerve pain. A large proportion of what were formerly called neuralgias

are due to neuritis, to some form of organic disease or to some reflex irritation. There is, however, a certain percentage of persistent nerve pains for which we can find no organic basis, and as a matter of both necessity and convenience we still retain the word. We qualify it according to its cause and its location. Hence we have hysterical, gouty and neuritic neuralgias, and we have trigeminal, cervical, brachial, etc., neuralgias, and we can speak of neuralgias as symptomatic or idiopathic according as we know or do not know the basis of the trouble. But there is little if any idiopathic neuralgia for the observant physician.

The most frequent form is the trigeminal; next in order come the brachial and sciatic.

Symptoms.—The dominant symptom is pain. This pain is sharp, darting, boring, stabbing or burning in character. It comes on in

	Strands of Cerebrospinal Nerves	Distribution
Area I.....	Trigeminus, facial, etc.	Face and its orifices, anterior scalp.
Area II.....	Upper four cervical.	Occipital region, neck.
Area III.....	Lower four cervical and first dorsal.	Upper extremities.
Area IV.....	Upper six dorsal.	Thoracic.
Area V.....	Lower six dorsal except last.	Abdominal wall, upper lumbar, upper lateral thigh surface.
Area VI.....	Twelfth dorsal, four lumbar.	Lumbar region, upper gluteal, anterior and inner thigh and knee.
Area VII.....	Fifth lumbar and five sacral.	Lower gluteal, posterior thigh, leg.

paroxysms of great intensity. In the intervals there may be no pain or it may be simply a dull ache. The pain runs along the course of certain nerves, though it is not confined necessarily to them, but may be somewhat diffuse.

The skin and nerves are sometimes tender or even exquisitely sensitive. Firm pressure, however, is usually not painful. In about half the cases of long standing, *tender points* may be found which correspond to the exit of nerves from a bony canal or the substance of a muscle or fascia. In rare cases there is tenderness over the spine corresponding to that point where the affected nerves arise. Besides feelings of pain, there is often a sense of numbness, cold, tingling, or heaviness of the limb. Vasomotor, secretory and trophic disturbances may occur; but when these are pronounced one must suspect neuritis or an organic central disease. Muscular spasm is sometimes present. The paroxysms of pain may intermit regularly; sometimes they come on every day at the same hour. They are apt to be worse at night. The attacks of the disease often run a course of several weeks or months, and in some forms they last for years.

The painful sensations of neuralgia usually originate in the peripheral sensory neuron. In some cases as in the neuralgia of tabes and perhaps sometimes in tic douloureaux and in the herpetic neuralgias, the posterior spinal ganglion is chiefly at fault; in others the irritation affects the entire sensory nerve. The central sensory neurons that take along impulses to the brain are rarely the cause of neuralgia, and local diseases of the cord and brain do not, as a rule, cause pain by irritating these sensory pathways. Still there may be neuralgic pain from this cause; and "central nervous pains" have been observed in brain tumors and after brain hemorrhage or softening, when these lesions have involved the posterior part of the optic thalamus.

Pathology.—Many cases that used to be called neuralgia are now known to be forms of neuritis or perineuritis; *e.g.*, sciatica and brachial neuralgia. Other forms are sometimes due to impaired nutrition of the neuron from an obliterating arteritis (tic douloureux); still others are due to the irritation of nerves from the diathetic poison of gout, rheumatism and diabetes, or to extrinsic poisons, such as alcohol, lead and arsenic. In these cases the sensory nerves of the nerve-sheaths (nervi nervorum) are affected. There remain many cases in which the trouble shifts from one locality to another or in which no special local or general irritation can be discovered. In these cases we assume that the pathogenic focus is in the spinal or cerebral sensory neurons and is a toxæmia or a psychosis.

There are some forms of neuralgia which may be called "reminiscent" or "hallucinatory." The patient, who is an impressionable and sensitive person, has had a genuine cause for neuralgic pains; but this, after lasting some time, has ceased, while the painful impressions continue to remain in the cerebral cortex. The neuralgia is a morbid habit of feeling pain. Such neuralgias are promoted often by the use of morphine. There are neuralgic pains occurring oftenest in the legs, but sometimes in the back, shoulder or arms, due to spasms of thickened arteries and causing pain just as the pain of angina pectoris is caused. These are called anginal neuralgias. They are often accompanied with cramping and weakness of the muscles.

Pathogenetically we can divide neuralgias into (1) toxic; (2) exhaustion; (3) neuritic and perineuritic; (4) anginal; (5) psychic. Often there is a combination of these factors.

Diagnosis.—This is based on the fact that neuralgic pains are shifting, paroxysmal, follow the course of nerves, are accompanied often by tender points and not accompanied by signs of organic nerve disease, such as paralysis, anæsthesia and tenderness over the nerve-trunk. Thermic sensations of burning or cold are rarely neuralgic, but are due to neuritis or some organic change.

The treatment will be discussed under special heads.

NEUROSES OF THE NERVES OF SPECIAL SENSE. THE OLFACTORY NERVE

Anatomy.—The olfactory nerves consist of a number of peripheral fibres that arise from the olfactory bulb, pass through the cribriform plate of the ethmoid bone, and are distributed to the mucous membrane of the superior and middle turbinated bones and the upper part of the nasal septum. The olfactory bulb is with its associated parts really a subdivision of the brain (rhinencephalon) and not a true nerve.

The central olfactory fibres pass to the hippocampus, cornu ammonis, and convolution of the corpus callosum. Through cells in these areas they are put in connection with the optic thalamus and with the motor tracts. The olfactory nerves do not decussate.

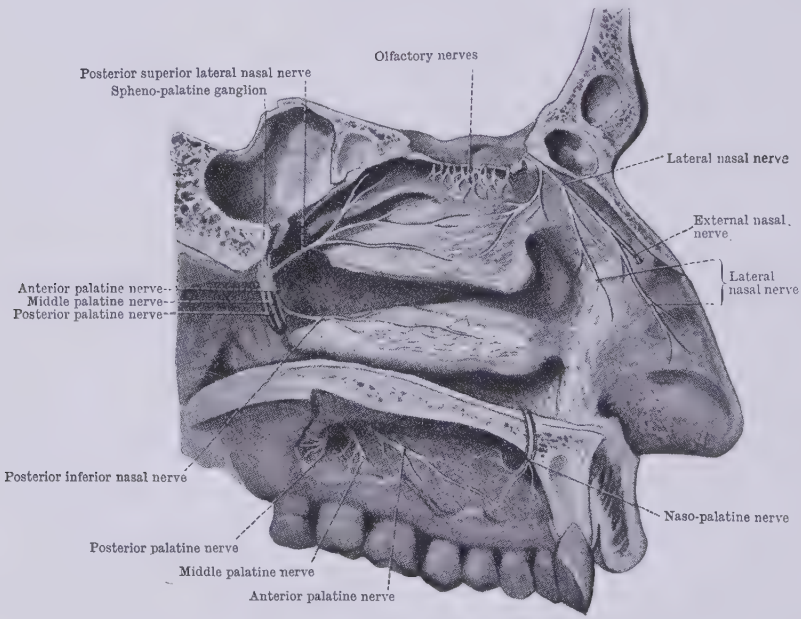


FIG. 68.—Showing distribution of olfactory and trigeminal nerves in nose. (*Cunningham.*)

The sense of smell is rudimentary in man, yet it is still the sense by which we can appreciate the most attenuated matter; for the trillionth of a grain of mercaptan is able to awaken a sensation in the mind, but has weight and dimensions so infinitely minute as to be quite beyond the power of the imagination to grasp. According to Valentin, we can percieve $\frac{1}{1200000}$ of a grain of oil of roses. According to Fischer and Penzoldt, one can perceive $\frac{1}{2760000000}$ of a grain of mercaptan. I have found that one can perceive the odor from 4 cm. of a solution of oil of cloves, 1 to 100,000. Matter to be perceived as odor must be in a gaseous form. Odorous sensations cooperating with taste sensations form "flavor." Variety in odorous sensations depends probably upon the rapidity of molecular vibrations as in the case of light;

and substances having similar relations in vibration have similarity in odor (Haycraft).

Anosmia.—The olfactory nerve is affected clinically by loss of function, or anosmia, increased sensitiveness of function, or hyperosmia, and perversions of function, or parosmia.

Anosmia is far the most common disorder of olfaction.

Etiology.—Its usual cause is disease of the mucous membrane of the nose. Injuries, inflammations and tumors affecting any part of the course of the nerve, its bulb or central fibers may also cause it. Unilateral cortical lesions in the uncinate gyrus may lead to partial

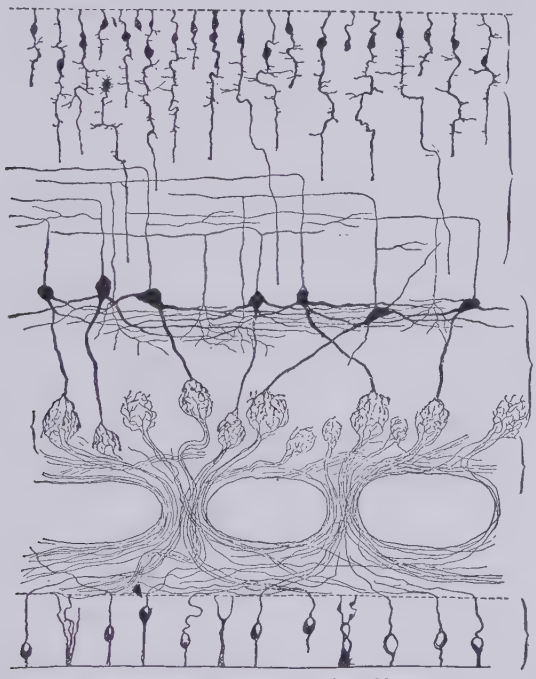


FIG. 69.—Principal constituent elements of the olfactory nerve of a mammal. (*Van Gehuchten.*)

loss of smell. It will be not entire, because each nerve receives fibres from both hemispheres. Paralysis of the fifth or seventh nerve may indirectly cause some anosmia. Primary degenerative changes due to syphilis of the nervous system, as in locomotor ataxia, and excessive olfactory stimulation cause anosmia. Anosmia occurs sometimes as a pure psychosis in hysteria, melancholia or in neurasthenic states. There may also be a congenital absence of the nerves.

Diagnosis.—This is made by test odors. To test the sense of smell, a bottle of oil of cloves or of some familiar non-irritating odor may be

used. To detect quantitative disturbance one may use six phials containing oil of cloves, in purity and in watery mixture of 1 to 10, 1 to 100, 1 to 1000, 1 to 10,000 and 1 to 100,000. Special olfactometers have been devised. The sense of smell for any single odor is lost in about three minutes, but returns after one minute's rest.

Treatment.—For functional anosmia, snuffs containing strychnine gr. $\frac{1}{30}$ and gum acacia ʒij can be used. Weak galvanic and faradic currents are recommended. Usually there is in anosmia a local diseased condition of the nose which requires treatment.

Hyperosmia occurs only rarely and then in neurasthenic, hysterical or insane persons. In the latter it is more often a psychical phenomenon than a peripheral disorder. Hyperosmia can be cultivated, and this is done sometimes by the blind and by those engaged in certain pursuits, such as tea tasting and wine tasting.

Hallucinations of smell occur in the insane, as just mentioned, and a few cases of epilepsy are reported in which the aura was a stench. In tumors of the temporal lobe irritating the olfactory cortical centre, "stench seizures" sometimes occur. When all olfactory sensations are disagreeable the condition is called kakosmia.

Parosmia is a not infrequent condition. In it everything smells alike to the patient, or a pleasant odor is perhaps a peculiar or offensive one. This condition may be due to local disease, but is often a symptom of hysteria or the psychoses.

THE OPTIC NERVE

Anatomy.—The optic nerve is not a true peripheral nerve, but a tract of the brain, and it connects the retinal cells with the brain proper. Like other brain tracts, its fibres have a myelin sheath, but no neurilemma. The real origin of the nerve is in the retina, just as the olfactory nerve arises in the peripheral cells of the olfactory mucous membrane and the spinal sensory nerves arise in the spinal ganglia.

The retina is a nervous tissue formed essentially of three layers of nerve-cells. From without inward they are: the layer of visual cells, the layer of bipolar cells and the layer of ganglionic cells. These different layers may be subdivided so as to give the following layers from without inward:

1. The layer of rods and cones. } Forming the layer of visual cells.
2. The external granular layer. }
3. The external molecular layer. } Forming the layer of bipolar cells.
4. Internal granular layer. }
5. Internal molecular layer. } Forming the layer of ganglion cells.
6. Ganglionic layer, with the fibres of the optic nerve. }

The layer of visual cells is subdivided, as seen in the figure, into that of the rods and cones externally and that of the external granular internally. This is, however, practically a layer made up simply of bipolar nerve-cells with prolongations more or

less long which run to the external surface of the retina and there form a series of bodies known as the rods and cones.

In the layer of bipolar cells are layers of cells with processes running horizontally, and in the internal molecular layer are larger horizontal cells, called by Cajal spongioblasts. There are also in the retina terminal arborizations of cells whose origin is in the thalamus, corpora geniculata, or anterior tubercles. These carry impulses *to* the retinal cells.

The neuraxons of the ganglionic cells send fibres which unite to form the optic nerve.

The optic nerves each contain about 500,000 fibres, which is about the same as the total of all the sensory fibres of the spinal nerves. They pass to the optic chiasm, where about one-third of the fibres cross, in man. In lower animals the decussation is greater. Those fibres which do not cross come from the outer or temporal third of the retina; those which do cross come from the internal or nasal two-thirds. A few fibres pass from one optic centre of the brain along the posterior border of the optic chiasm to the centre of the opposite side (commissure of Gudden). After leaving the chiasm, the fibres form the *optic tract*. The tract curves up and back around the crus cerebri, and divides into a lateral and mesial root.

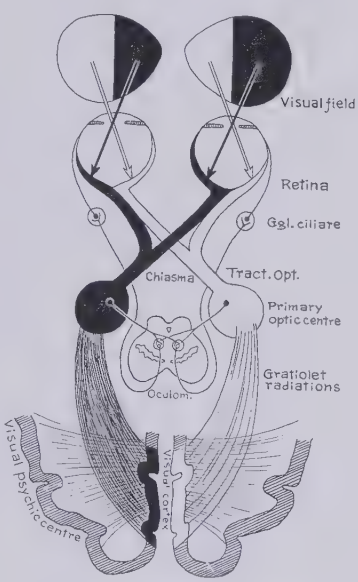


FIG. 70.—Showing relation of occipital cortex and its visual centres to the eye and visual fields. (*Bing.*)

These roots connect with the external geniculate body, the anterior tubercles of the corpora quadrigemina and the posterior ganglion of the thalamus, *i.e.*, the pulvinar. These ganglia are called the primary optic centres. Through the anterior tubercles of the corpora quadrigemina and by other means, the optic nerve is connected with the oculo-motor nerve, and thus reflex movements of the pupils, lids and eyeballs are brought about.

From these primary optic centres, fibres enter the posterior part of the internal capsule, curve up and back toward the occipital lobes, forming the optic radiations of Gratiolet. The inferior longitudinal bundle is part of these radiations and connects the external geniculate body with the calcarine cortex (A. Meyer).

They are finally distributed to the cortex of the occipital lobe, and in man chiefly to the cuneus and the parts about the calcarine fissure.

It will be seen that each retina is connected with the occipital lobe of both hemispheres; further, that the outer or temporal half of each retina is connected with the occipital lobe of the same hemisphere, and the inner or nasal half of each retina with the occipital lobe of the opposite side. The upper part of each retina seems to be connected with the lower part of the cuneus, and *vice versa* (Fig. 70).

Other associations exist by which the optic centres on the two sides are connected with each other and with other cranial nerves in the medulla.

The optic nerve is a nerve of special sense of vision and has no other function except that of an excito-reflex character.

DISEASES OF THE OPTIC NERVE

The optic nerve may be affected by nearly all forms of pathological change. For the neurologist, however, the especially important conditions are inflammations, degenerations, injuries and functional disorders. Inflammation of the nerve, or optic neuritis, may occur as a papillitis or inflammation of the head of the nerve, a neuro-retinitis or descending neuritis, or a retro-bulbar neuritis. Perineuritis is rare. Neuro-retinitis and papillitis are closely associated clinically and pathologically (Noyes), so that practically only two forms of neuritis need be discussed separately.

Papillitis and Neuro-retinitis—Etiology.—This condition is seen by

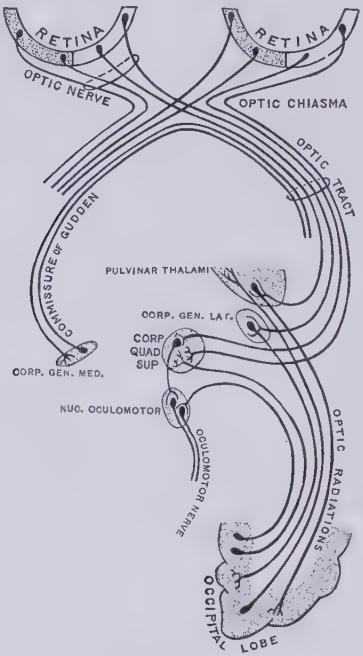


FIG. 71.—The optic nerve showing detail of the primary optic centers. (*Cunningham.*)

neurologists in connection with brain tumors, brain abscess, meningitis and occasionally multiple neuritis. The other causes are nephritis, diabetes, infectious fevers, lead, and severe hemorrhages. The disease occurs at all ages and in both sexes. In brain tumors it occurs in two-thirds of the cases, and especially often in cerebellar tumors. It occurs in 80 per cent. of cases of tuberculous meningitis.

Symptoms.—Subjective symptoms are often not present. The vision may remain good for a long time. In other cases there are concentric limitation of the vision field, loss of color sense, and scotomata.

For a description of opthalmoscopic changes the reader is referred to a special text-book. It is in this condition that "choked disc," which is a papillitis with much serous infiltration, occurs. The changes are less striking in neuro-retinitis. The disease may affect one or both nerves. In brain disease both nerves are usually involved.

Pathology and Pathological Anatomy.—The process is usually subacute or chronic. Congestion, exudation, small hemorrhages and collections of leukocytes occur. The sheath of the nerve just back of the globe is often distended with a serous exudate. After a time the nerve-fibres atrophy, connective tissue proliferates and takes their place, and we have a secondary optic atrophy.

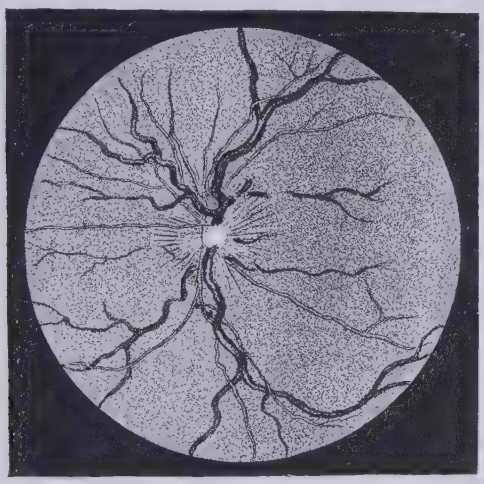


FIG. 72.—Neuro-retinitis. (*Jaeger.*)

The process is essentially peripheral, but it extends back with lessening intensity into the trunk of the nerve. The purely mechanical theory of neuritis, that it is due to compression, can be accepted only in some cases. It is probable that the neuritis results from an irritating serous fluid which extends down the sheath of the nerve, this sheath being a prolongation of the arachnoid cavity. Mechanical causes lead to constriction, accumulation of the fluid, and compression of the nerve at its periphery, and hence to inflammation. Sometimes, at least, the irritating fluid contains microbes or microbic poisons. (See also Brain Tumors.)

Retrobulbar Neuritis.—In this disease the lesion lies chiefly behind the globe. Its causes are especially toxæmia from alcohol and tobacco. It is also due to rheumatic toxins, syphilis, lead and diabetes.

In the acute cases there is usually rather rapid loss of sight, with some pain and tenderness. The ophthalmoscopic changes are relatively

slight. In chronic cases, which are usually toxic in origin and due to alcohol or tobacco, or oftenest to both; there are color scotomata or absolute scotomata and amblyopia. There is no pain. The condition is known as tobacco or alcoholic amblyopia.

The *prognosis* of neuritis varies with the cause. If this is removable, as in the toxæmias, recovery is the rule. This is a proof that in neuritis the connective tissue is the part chiefly involved, for a destroyed or atrophied optic nerve does not recover. In many cases, however, atrophy follows the neuritis.

The *treatment* is based on the cause. One may need to use cups, salicylates, the iodides and mercury and salvarsan; later, the iodides and strychnine. The rhinologist and surgeon may be needed.

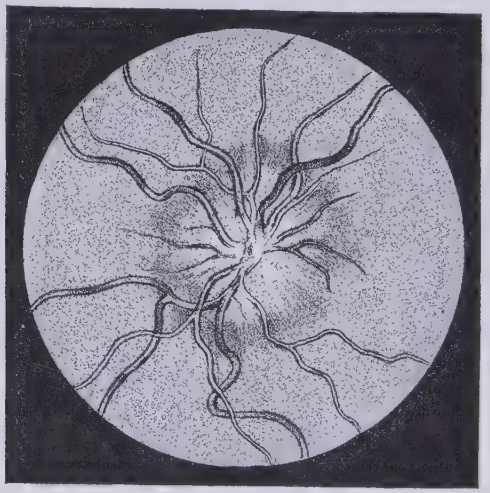


Fig. 73.—"Choked disc."

Degeneration of the Optic Nerve, or Optic Atrophy.—This condition may be primary or secondary. Secondary atrophy is usually the results of a neuritis. I shall describe here *primary optic atrophy.*

Etiology.—It occurs oftener in men than in women (three to one). It may be hereditary; it may be the result of some toxin; but practically, it is usually due to syphilis and appears in tabes or paresis. It occurs in multiple sclerosis and hereditary ataxia. It may occur apparently as a simple primary affection, though these cases are generally optic forms of tabes dorsalis.

The *symptoms* are those of gradual decrease of acuity of vision, concentric limitation of the visual field, loss of color sense, dilatation and immobility of the pupil. The sense of sight may remain good for a long time. Ophthalmoscopically, the nerve disc is opaque, grayish, or dirty-

looking, and often has a cup-shaped or "cupped-disc" appearance. The vessels are smaller and few in number.

The *pathology* and *pathological anatomy* are that of a parenchymatous degeneration with loss of nerve-fibres, which are replaced by connective tissue.

The *prognosis* is almost uniformly bad.

The *treatment* is that usually of the cerebral or spinal disease. Mercury, iodides, strychnine, phosphorus and iron are given. Warm baths and salicylate of soda sometimes have a temporarily good effect. Strychnine in physiological doses gives, however, the best symptomatic results. Electricity is not of any use. Stretching the nerve does no good. Injections of salvarsan have sometimes been efficient.

The optic nerves and their primary and cortical centres are subject to various other diseases. So far as these are organic, they will be described in detail under the head of brain diseases. But there are certain symptoms often of functional origin which are best described here. These are: (1) Amblyopia and amaurosis; (2) retinal hyperæsthesia and dysæsthesia; (3) hemianopsia.

Amblyopia and Amaurosis.—Amblyopia is a partial loss or dimness of vision, there being no observable lesion of the eye, or its nerves. Amaurosis is a total loss of vision, also without observable lesion.

The causes are shocks, hysteria, migraine, concussion of the brain, and severe hemorrhages. There are also certain toxic causes, chiefly alcohol and tobacco, quinine, and salicylic acid. Other causes are arterial sclerosis and renal disease leading to spasm of the retinal vessels or of those of the occipital cortex. Night blindness and snow blindness are forms of functional amblyopia.

The *symptoms* are diminution or loss of vision, usually sudden, temporary and involving both eyes. Amblyopia in hysteria is usually greater in one eye and associated with concentric limitation of the visual field and disturbance of color sense.

Underlying amblyopia there may be minute hemorrhages in the brain, causing temporary pressure, or a vascular spasm, causing anæmia.

Retinal or ocular hyperæsthesia is a condition in which the eye is abnormally sensitive to light. It may be due to exposure to extreme light or to seclusion in a dark room. The neurologist sees it oftenest as a symptom of hysteria (*vide* Hysteria) and neurasthenia. It occurs in mydriasis and albinism. It is not to be confounded with photophobia due to irritation of the conjunctiva.

Nyctalopia, or the condition of seeing better in a dim light, is a form of the disease.

Hemianopsia, or half-sightedness, or hemianopia, a condition in which there is a blindness of one-half the visual field, may be due to a

functional or organic disorder of the nerve or its centres. It is a symptom of many lesions and conditions, and can be described only generally here.

Etiology.—Its principal functional cause is migraine. Its organic causes are tumors, inflammations, softenings or hemorrhages involving part of the optic nerve or its central connections.

Symptoms.—Various descriptive terms are used to indicate the character of the hemianopsia. In lateral hemianopsia a vertical half of the field is involved. In lateral homonymous hemianopsia there is half-blindness on the left or right side of each eye, as the case may be. In temporal hemianopsia the outer halves of the eyes, and in nasal the inner halves, are involved. The upper or lower segments or irregular segments of the visual field may be involved.

These various forms of hemianopsia depend upon the location of the lesion which cuts into and destroys the optic fibres in their course from the eye to the visual centre in the occipital cortex. The mechanism will be understood when it is remembered that each occipital lobe is supplied by nerve-fibres from one-half of the retina of each eye. A cut shows this better than any description (Figs. 70, 71). In binasal hemianopsia the lesion must be in front of or practically directly on the chiasm. In bilateral temporal hemianopsia it must be double and at each side. In lateral hemianopsia the lesion must lie farther back than the chiasm, in the tract, the primary centres, the optic radiations or occipital lobes.

In hemianopsia from disease of the nerve as far back as and including the primary centres in the optic thalamus and corpora quadrigemina there is a loss of light reflex when a ray of light is thrown upon the blind side of the retina, but the pupil still contracts when light is thrown on the sensitive side of the retina. This phenomenon is called "Wernicke's hemiopic pupillary reaction." If in hemianopsia the light reflex is preserved, the lesion is back of the primary centres and involves the optic radiations or cortex. The test can only be made with special apparatus.

A test for the condition of hemianopsia in its early stage, and one that is useful in stupid or partially comatose patients, is the following: When the finger is suddenly brought in front of the eye on the sound side, there is a wink; if brought in front from the blind side, the orbicularis does not contract.

Hemianopsia is almost always the sign of organic disease. It is not found in hysteria, but does occur in migraine. It is best made out and recorded by means of the perimeter.

Its course and treatment depend upon the cause.

SENSORY NEUROSES OF THE TRIGEMINAL NERVE

Anatomy.—The trigeminus or fifth nerve is one of the most extensively distributed and most delicately sensitive nerves of the body. Its sensory branches represent the atrophied and lost sensory roots of the third, fourth, sixth, seventh and twelfth cranial nerves. The trigeminal nerve is a mixed nerve. It has two nuclei of origin; a central nucleus for the motor part and a peripheral nucleus for the sensory part. The motor nucleus has two parts: a chief nucleus lying deeply in the substance of the pons Varolii, and an accessory nucleus, which consists of a long tract of gray matter, known as the descending root and lying in the upper part of the dorsal portion of the pons. It passes down along the side of the aqueduct of Sylvius. The sensory root of the trigeminal has its origin in the Gasserian ganglion, which is composed of unipolar cells, like those of the spinal ganglia. The axis-cylinder processes of these cells bifurcate; the external branches pass outward and become part of the peripheral sensory nerve, the

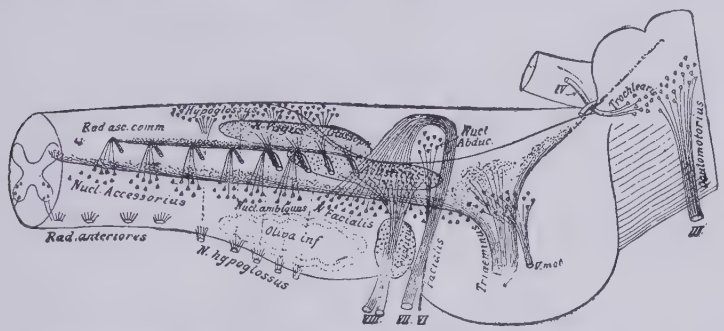


FIG. 74.—Showing the origin of the cranial nerves and the extent of the origin of the trigeminus. (*Edinger.*)

internal branches pass into the substance of the pons, and there give off ascending and descending branches. The ascending branches are short, and pass to a terminal nucleus, known heretofore as the sensory nucleus of the trigeminus. The descending branches pass down to the pons and medulla, as far as the cervical part of the spinal cord and form the *ascending root* of the trigeminus.

The nuclei of the trigeminus reach the whole length of the pons and medulla, and are co-extensive with the origin of all the other cranial nerves (Fig. 74). Hence the frequency with which its disorders are complicated with those of these nerves. Its cortical origin is probably in the lower part of the post-central convolution.

The trigeminus supplies sensation to the face, conjunctivæ, nose, the frontal and maxillary sinuses, the teeth, the palate, tongue and part of the upper pharynx; also to the scalp as far back as the vertex and to the external auditory meatus (Fig. 75). The distribution is not always the same and is helped by fibres from the cervical nerves.

It gives sensation also to the anterior three-fourths of the dura mater, the falx and probably the tentorium. The pia and arachnoid are not sensitive. The posterior fossa and the occipital part of the dura mater are supplied by the vagus. The trigeminus also supplies the above-named parts with trophic, vasomotor and secretory fibres. The vasomotor fibres are brought to it, in part, from the medulla and cervical spinal cord *via* the sympathetic; the secretory fibres have the same origin. An exception is to be made of the lachrymal secretory fibres which are brought by the motor nerves of the eyeball. The opinion, based largely on physiological experiment, that

the trigeminus sends trophic fibres to the conjunctivæ and cornea is apparently contradicted by the many successful cases of entire extirpation of the Gasserian ganglion without any inflammation of the eye following.

The trigeminus supplies motion to the muscles of mastication, viz., the two pterygoids, the temporal, masseter, mylo-hyoid and anterior belly of the digastric. The sensory neuroses of this nerve are neuralgia, paræsthesia and anæsthesia.

Neuralgias of the Trigeminus.—The trigeminal nerve is subject to two types of neuralgia, viz.: (1) The symptomatic pains; (2) tic douloureux.

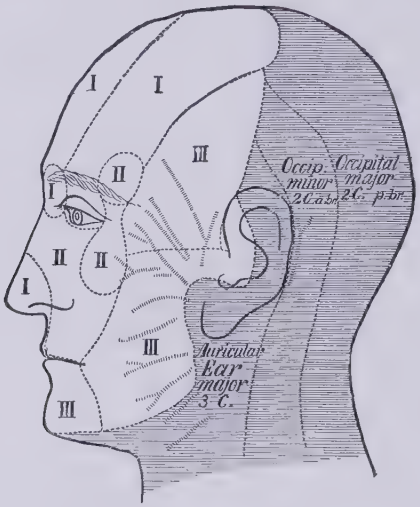


FIG. 75.—Showing the distribution of the sensory nerves of the face. *I, II, III,* First, second, and third branches of the fifth. The shaded part is supplied by the cervical nerves; 2 *C.a.br.*, second cervical anterior branch; 2 *C.p.br.*, second cervical posterior branch.

1. The *symptomatic pains* are by far the most frequent.

They may be supra-orbital, infra-orbital or supra-maxillary, infra-maxillary or dental, and mixed forms. The most common type is the supra-orbital; next, the mixed form.

Etiology.—The female sex is oftenest affected; most cases are seen in the first half of life; most attacks occur in the winter and spring. The left side is oftener affected. The second and third branches of the fifth nerve are most susceptible to rheumatic influences, the first branch to malarial and septic poisons. Dental disorders naturally are frequent factors in neuralgia of the second and third branches. Exposure, infections and other depressing influences are factors in causing these neuralgias. Ocular, nasal, antrum and frontal sinus disease may cause pain in the supra-orbital nerve. Syphilis, malaria, hysteria,

epilepsy, trauma, rheumatism, tumors, bony disease all may be factors in causing trigeminal pains. It is sometimes due to herpes zoster.

Symptoms.—The pains of trigeminal irritation are sharp and intense, with exacerbations and remissions. Their character and duration depend on the cause. There is no specific disease to describe (see migraine and headache).

Tic Douloureux (*Prosopalgia, Fothergill's Neuralgia, Epileptiform Neuralgia*).—Tic douloureux is a special form of trigeminal neuralgia occurring in middle or advanced life, unusually severe in its symptoms and obstinate in its course. It ought to be distinguished sharply from the ordinary forms of trigeminal neuralgia. These latter are symptomatic pains almost altogether; while tic douloureux is a special disorder, dependent upon changes in the nerve itself.

Etiology.—It occurs, as a rule, in persons who are over forty, and is seen in the very aged. It is, indeed, almost the only neuralgia which old people have. It occurs in men and women in about equal frequency. It is brought on by exposure, overwork and depressing influences; sometimes, perhaps, by local diseases of the teeth, antrum and jaws. There is almost always decided evidence of arterial sclerosis.

Symptoms.—It is characterized by intense darting pains, which usually start in the upper lip and by the side of the nose. From here they radiate through the teeth or into the eye and over the temple, brow and head. They are confined to one side of the head. During a paroxysm the face usually flushes, the eyes water, the nose runs and the patient assumes an expression of the greatest agony. The attack lasts for a few minutes, then becomes somewhat less, and the pain may cease entirely. A breath of cold air, speaking, eating, putting out the tongue—all bring on paroxysms. The pains are worse in winter and often become less or cease during summer. Occasionally they come on for a few months every year, usually during the spring. The pains are always limited to one side of the face and are centred chiefly in one branch of the nerve oftenest in the supra-maxillary and next in the infra-maxillary. They may spread so as to involve the whole of one side of the face and tongue.

Spasmodic movements of the face, tongue or jaws may be associated with the pain.

Examination rarely reveals any objective trouble, but in a few cases some anæsthesia may be noted. The disease causes loss of sleep and impairment of nutrition through the pain caused by eating. Much depression of spirits naturally exists, for the pain is the worst agony that nature in a hellish mood could devise. There is a tendency to a cure, however, and some patients get well spontaneously in from seven to fifteen years.

Pathology.—The disease is usually a degenerative one, and probably is due to irritative and atrophic processes occurring in the nerve and its ganglion. A low grade of neuritis, perhaps from alveolar disease, has been found sometimes, but as a rule the nerve does not appear much changed. The arteries supplying the nerve, however, often undergo the changes of endarteritis, their calibre is much lessened, and the nerve cannot get its proper supply of blood. Thus an obliterative arteritis underlies some cases of the disease. The ganglion shows sometimes evidences of degenerative changes, but these must usually be considered secondary, for division of the nerve almost invariably stops the pain for one or two years at least. On the whole, the most satisfactory view of the pathology as, I first showed, is that the neuralgia is due to an obliterating neuritis causing a defective nutrition of the nerve and accompanied with vascular spasm. *Tic douloureux is a kind of trigeminal angina.*

Some cases occurring in younger people are explained by the presence of distinct local disease and a hysterical pain habit.

Treatment.—In cases which occur in old people, the use of nitroglycerin given in doses of gr. $\frac{1}{200}$ q. 2 h. sometimes has a happy effect. An occasionally good remedy is crystalline aconitia given in doses of $\frac{1}{200}$ gr. until its physiological effect is obtained. Quinine, gelsemium, croton chloral and codeine may be found useful. The common practice of pulling out all the teeth is almost always unsuccessful, and ought not to be undertaken without specially good reason. Tonics containing iron, phosphorus, quinine or arsenic are generally helpful. In younger patients the remedies recommended under the head of migraine and headache may be given. Change to a warm, equable climate may be tried; it is not a certain resource.

I have found that in cases not of over four or five years' duration, rest in bed with massive doses of strychnine often effects striking cures. The drug should be given hypodermatically in doses of gr. $\frac{1}{30}$ once, or better three times, daily, gradually and very slowly increased until gr. $\frac{1}{6}$ or $\frac{1}{5}$ three times a day is reached. This is repeated four days and then the amount gradually reduced. The whole treatment takes about six weeks. The patient must be kept rigidly quiet in bed or in the bed-room and the full course persisted in. After finishing the strychnia, iodide of potassium and iron are given. The treatment may have to be repeated with lessened rigor. The treatment by large doses of opium, gr. iii. to vi. daily, is uncertain and often dangerous. Sometimes massive doses of quinine break up an attack.

Finally, surgical interference may be necessary. The removal of the nerve at as deep a point as possible is the only operation to be seriously entertained. This sometimes causes cure, but, as a rule, the pain comes

back in six months to a few years. Even such a respite, however, is often gladly seized upon. Removal of the Gasserian ganglion has been attempted with success. There are numerous methods of operating upon the different branches of the trigeminus and resecting it distal to the ganglia.

Hartley, of New York, has devised an operation by which he enters the middle fossa through an opening in the temporal bone, thus reaching the root of the nerve. Abbe has modified it very successfully. He trephines the skull, enters the middle fossa, cuts off the second and third nerve-roots and plugs the foramen ovale. The operation in proper hands is safe and most efficient. Injection of the Gasserian ganglion with alcohol introduced by a needle passed through the cheek and upward behind the jaw through the foramen ovale is an operation which has been successful. Injections of alcohol are also made into the different branches of the nerve. The treatment is a safe one and produces often temporary good results. On the whole it is a rather disappointing procedure.

Trigeminal Paræsthesia.—Sometimes persons suffer from peculiar numbness, thrilling or formication in the course of the trigeminus. The sensation may be nearly constant and excessively annoying. It never amounts to actual pain. It occurs in anæmic, nervous and hysterical persons. It may follow an attack of herpetic neuralgia of the face. It is to be regarded as an abortive form of neuralgia or neuritis and so treated.

Trigeminal Anæsthesia.—This occurs from various pathological lesions in the course of the nerve or in its nuclei. The most common organic causes are brain tumors, syphilitic disease of the membranes at the base of the brain, and herpes. Trigeminal anæsthesia occurs together with anæsthesia of other areas in hysteria and in organic disease of the nerve-centers. It is sometimes noted in tic douloureux and facial hemiatrophy.

Flushing, pallor, lacrymation, salivation, are all symptoms of disturbance of the trophic, vasomotor and secretory fibres running in the trigeminal nerve. They are usually, if pathological, only concomitant symptoms of other diseases.

Facial herpes may occur due to an inflammation of the Gasserian ganglion, and it is accompanied with much pain followed by cutaneous anæsthesia and paræsthesia, but it is a malady generally belonging to internal medicine or dermatology.

HEADACHE (CEPHALALGIA)

Headache is the name given to attacks of diffuse pain affecting different parts of the head and not confined to the tract of a particular

nerve. It usually comes on in paroxysms at various intervals, but may be continuous.

Etiology.—Headache is the most common of nervous symptoms. Ten to fifteen per cent. of school-children, twenty-five per cent. of men, and over fifty per cent. of women are subject to it, more or less, though this proportion would be much reduced if migraine were excluded from statistics.

The headache ages are from ten to twenty-five and thirty-five to forty-five; most cases occur between the ages of eight and twenty-five, especially in females. The number of headaches increases gradually from the period five to ten years up to the period fifteen to twenty, then falls until the thirty-fifth year, and rises again until about the age of forty. Early childhood and declining age are practically exempt from chronic functional headaches. Women suffer from it more than men in the proportion of about three to one. It is more frequent in city populations and among the wealthier classes. Headaches are more common in the spring and fall and in temperate climates. Headaches may be classed, in accordance with their causes, as follows:

1. Exogenous and endogenous toxæmias, in which impoverished or disordered blood is brought to the brain, as in (*a*) gastro-intestinal disorders; (*b*) diathetic states: gout, rheumatism, uræmia; (*c*) infections: malaria, fevers.

2. Toxic causes: lead, alcohol, tobacco, etc.

3. Neuropathic states: epilepsy, neurasthenia, hysteria.

4. Reflex causes: ocular, nasopharyngeal, auditory, gastric, sexual.

5. Organic disease, including arteriosclerosis, syphilis, tumors, neuritis, meningitis and diseases of the cranial bones. Very frequently several causes act together. The autotoxic, dyspeptic, ocular and neurasthenic are the common forms of chronic and recurrent headache.

Edinger thinks that a large percentage of chronic headaches is due to the presence of certain nodules of rheumatic or myositic origin situated near the origin of the muscles of the back of the head. Massage by removing these relieves the headache. I have not found these often, and doubt if they have so much importance, but their existence and possible influence should be borne in mind, especially in gouty subjects.

Symptomatology.—Headaches may be classed in accordance with their location and the character of the pain. We have accordingly: (1) frontal headaches; (2) occipital headaches; (3) parietal and temporal headaches; (4) vertical headaches; (5) diffuse headaches and various combinations of the above.

The most common form of headache is the frontal, next the fronto-occipital or diffuse, next the occipital.

The kind of pain differs with different persons and with different causes. We have: (1) pulsating, throbbing headache; (2) dull, heavy headache; (3) constrictive, squeezing, pressing headache; (4) hot, burning, sore sensations; (5) sharp, boring pains.

The first form characterizes headaches with vasomotor disturbances, and usually indicates migraine (throbbing headache). The second is usually of a toxic or dyspeptic type (the dopey headache). The third is found in the neurotic and neurasthenic (the pressure headache). The fourth in rheumatic and anæmic cases. The fifth in hysterical, neurotic and epileptic cases.

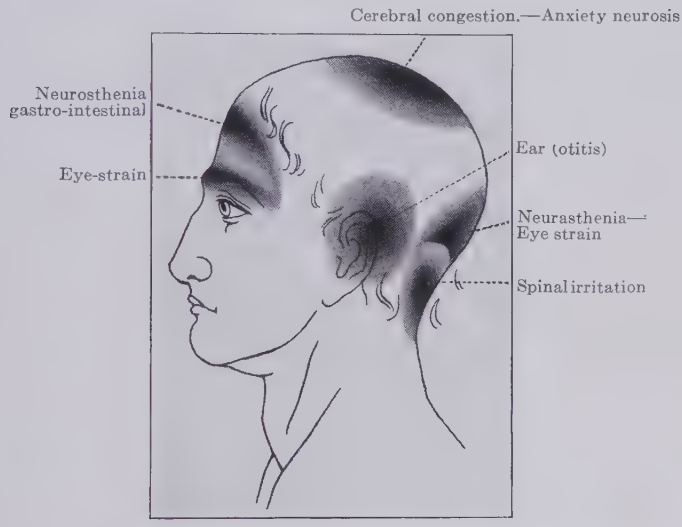


FIG. 76.—Reflex and symptomatic head pains.

The accompanying diagram shows some of the relations of localized pain to the cause. Chronic occipito-frontal headaches are usually neurasthenic, or ocular or both. Chronic occipital headache is usually neurasthenic, ocular or uterine.

Headaches may continue for a day or may last for weeks or months. Some persons have headaches only when constipated or bilious or when they have an attack of indigestion. Others suffer from a little pain nearly all the time, exacerbations occurring at various periods. Neurasthenic and ocular headaches are generally of this type. When headaches are persistent, examination should be made of the eyes of the nose and sinuses; the patient should be questioned as to syphilis, the continuous use of tobacco, of tea and coffee and of chronic dyspepsia. The possibility of brain tumor, of pachymeningitis from blows, or sunstroke or chronic alcoholism should be considered.

The persistent headaches not relieved by ordinary treatment are due to eye trouble, neurasthenia psychasthenia, rheumatic nodules, syphilis or pachymeningitis. Eye-strain may cause true migraine or ordinary headache. Eye-strain headache is usually associated with some weakness of eyesight and pains and discomfort about the globe, besides severer pains at times in the brow or occiput. The cause of the eye-strain is usually astigmatism and hypermetropia. Occasionally it is due to weakness or lack of balance of the eye-muscles.

Headaches may occur regularly every morning on awaking. They are called *morning headaches*, and are a symptom of neurasthenia, gastro-intestinal toxins or arterial sclerosis. These occur oftenest in middle life or later.

Symptoms Associated with Headache.—The symptoms oftenest associated with chronic and recurrent headaches are vertigo, somnolence, sensations of heat and pressure (cerebral paræthesias), and nausea. Vertigo goes oftenest with headaches of dyspeptic origin; some of the so-called bilious headaches of early life develop later into attacks of vertigo. This symptom often occurs with frontal headaches.

Pathology.—Headaches are to be distinguished from neuralgias and from a special and common form of head pain known as migraine.

Headaches are diffused pains caused, as a rule, by irritations located in or referred to the peripheral ends of the fifth nerve supplying the dura mater.

Neuralgias, on the other hand, are caused by irritations of the ganglia or trunks of these nerves. The pains are local and confined to the single branches of the nerve.

Migraine is a periodical neurosis in which there is a nervous discharge of force, not only affecting the trigeminus, but often other cranial nerves as well as sympathetic fibres. It is a general disease of which the headache is only one symptom.

The nerves of the dura mater are those most involved in headache. Headaches, when occipital, involve the sensory fibres of the vagus and the upper four cervical nerves. There is no anatomical change in the nerves except in organic headaches. But in many cases the membranes of the brain and their sensory nerves are congested or anæmic.

Diagnosis.—No symptom requires more careful investigation as to its cause than that of headache; for the diagnosis is always to be made, not of this symptom, but of its cause. Most of the foregoing description accordingly refers to etiology.

Headaches which persist for months, are worse in the day and leave the patient able to sleep at night, to recur on waking, are exhaustion pains and are due to a neurasthenic state.

Chronic headaches, worse at night, are usually of specific or organic origin.

Migrainous or "sick-headache" comes on paroxysmally, lasts a short time, and then leaves the patient feeling perfectly well or even better than ever. The attacks are often accompanied with nausea, flashes of light, strong pulsations of the head, vertigo, pallor or, more rarely, congestion of the face.

In neuralgic headaches the pains are sharp and shooting; they run along the tract of the nerve, and often are associated with suffusion of the eye and œdema. Tender points are felt.

Most of the persistent, recurrent headaches are of migrainous origin.

Treatment.—The constitutional treatment is based upon the etiology. Tonic measures, regulation of diet, securing a regular movement of the bowels, attention to ocular troubles, abstention from tobacco and alcohol and overwork are the important measures.

The symptomatic treatment consists in giving sedative drugs and local anodynes. The coal-tar products, antipyrin, pyramidon, antifebrin, are the best symptomatic remedies.

Antipyrin can be given in doses of gr. v. every twenty minutes until three or four doses are taken. Phenacetin often needs to be given in large doses of ten or even twenty grains. Antifebrin must be given in small doses. Combinations of caffeine citrate and the various coal-tar products furnish the basis for the ordinary headache cures. Local applications of a 20 per cent. solution of menthol, the ice bag, cloths wrung out in hot water or a piece of sheet lint soaked in chloroform liniment two parts and tincture of aconite one part are efficacious measures. A cathartic, rest in a darkened room, light diet—all these are measures which many patients themselves learn to adopt.

In headaches from organic disease, we have to resort to iodide of potassium, mercury, arsenic, salvarsan and the use of some preparation of opium.

As will be seen, each case of headache requires special treatment and a certain amount of experimentation in order to learn the idiosyncrasy of the patient.

MIGRAINE (SICK-HEADACHE, HEMICRANIA)

Migraine is a constitutional neurosis characterized by periodical attacks of pain chiefly in the course of the fifth nerve. The pain is often associated with nausea or vomiting, mental depression, vasomotor disturbances, such as flushing, or pallor of the face, by flashes of light, vertigo, tinnitus aurium, and in rare cases by partial paralysis of one oculomotor nerve.

It will thus be seen that migraine is more than ordinary headache and unlike an ordinary neuralgia.

Etiology.—The disease is very common in civilized countries and is frequent in America. It occurs oftener in women than men in the proportion of about three to one, and it begins in most cases at or a little before the age of puberty. It may begin as early as the fifth or even the second year. It occurs in neurotic families, and there is very often a history of direct inheritance. Other neuralgic troubles, epilepsy and gout may be found in the family history. The attacks occur oftenest in the winter in our climate. The cases that begin in childhood and early life are sometimes started by overwork at school, but usually no especial cause can be found. When they begin after maturity, a history of excesses in work, injury, shock or exhausting disease is found. Migrainous patients often have some refractive disorder of the eye or a weakness of eye-muscles, and these conditions may be factors in bringing on or keeping up the headaches. Endogenous toxemia and especially poisons developed in the intestinal tract are an important factor in migraine.

Symptoms.—The patient for several days may feel a sense of malaise and depression; usually, however, the prodromal stage lasts only a few hours or a day. The attack often comes on in the morning and gradually increases in intensity until the victim has to give up work and lie down. Sometimes the pain comes on with almost epileptic suddenness and violence, waking a person from sleep or compelling him at once to lie down. *Fulgurating migraine* is the term applied to this type.

The pain starts in one side of the head, usually in the temple and eye, but often in the occiput. It increases and finally may involve the whole head. The pain is of a tense, throbbing character, increased by jars, light and noises. It is accompanied sometimes by dimness of vision, often by flashes of light or by dark or light spots, variously colored, floating before the eyes (Fig. 77). Contraction of the visual field, usually in the form of bilateral hemianopsia, may occur. Vertigo, tinnitus aurium, confusion of ideas, feeling of stupor, disturbances of memory, are not uncommon; nausea and even vomiting are the rule. The vomited matter is at first chiefly mucus, but it may later become yellow and bitter from the presence of bile. Hence the term "bilious headache," which is an improper one, because the bile is only the result of retrostalsic action from the vomiting. Migraine is not the result of gastric or liver disorder.

The patient's face usually is pale and gives the evidence of acute suffering. The flushed face is very rare; the distinction between angiospastic or pallid migraine and angioparalytic or congestive migraine is not of clinical value. The blood pressure may rise and the pulse be lessened in rapidity. The temperature in children often rises.

The attack lasts from six to twelve or twenty-four hours, occasionally

even two or three days. As the intensity of the pain lessens, the patient sinks to sleep, and awakens next morning feeling refreshed and better than before the attack.

The attacks occur at varying periods, fortnightly or monthly, and even weekly. In women they often occur during menstruation. Some women are entirely free from them during pregnancy. At about the time of the menopause in women, and at about the same time of life in

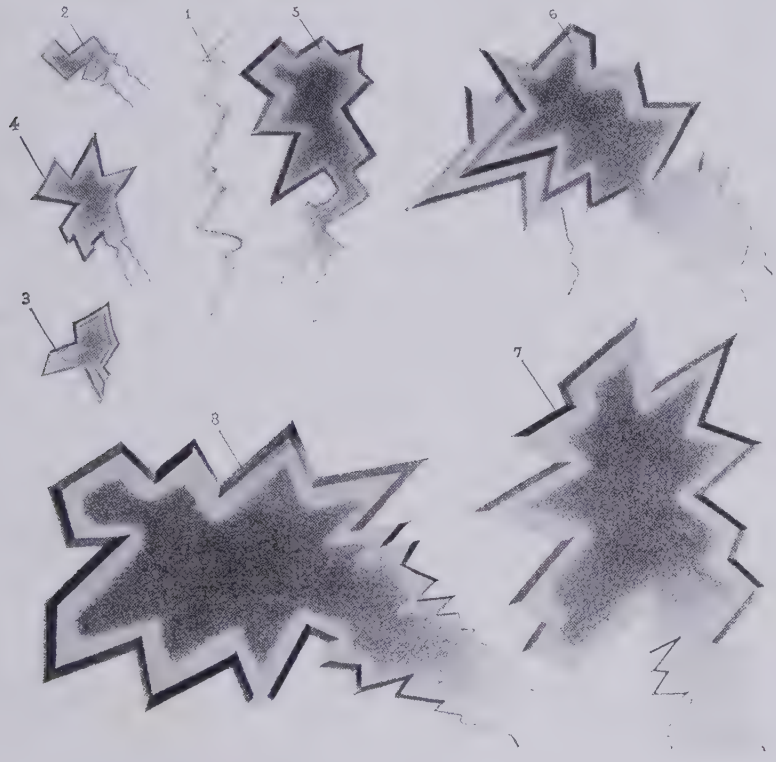


FIG. 77.—Scintillating and zig-zag lights seen in migraine. (*Babinski.*)

men, the disease lessens in severity and, as a rule, disappears. Some form of neuralgia or some neurosis in rare cases takes its place.

Complicating Symptoms.—Partial oculomotor paralysis, temporary aphasia, slight hemiplegia, heminumbness, peculiar odors or tastes, convulsive movements of the body almost resembling epileptic attacks occur.

Cases presenting these symptoms are rare. When they occur in one case, however, they usually occur in each attack unless it is modified by treatment.

Vicarious Attacks.—Migraine is sometimes associated with epilepsy or insanity; that is to say, persons in early life have had migraine and later developed the diseases mentioned. The relation between these diseases, is not, however, a very close one, nor does one disease lead to the other. The attack of migraine is sometimes replaced by various other functional neuroses. Sometimes, instead of a fully developed attack, the patient has a sense of mental depression, with confusion of ideas. Cases have been reported in which acute mania took the place of the headache.

Types.—Practically, we find two classes of cases:

1. The typical, associated with visual disorders and having most of the symptoms described above.

2. The irregular or mixed type, in which, with many symptoms of ordinary migraine, there is a history of rheumatic influences and often of anæmia or dyspepsia. These are cases of a true migrainous affection complicated with some form of symptomatic headache, such as has been already described. The mixed or irregular migraines are important to recognize, for they call for special treatment. Many patients have their "sick-headaches" and their "neuralgic headaches," so-called, and they distinguish between them. Both are migrainous headaches, but with other elements entering.

Pathology.—The seat of the pain is chiefly in the intracranial branches of the fifth nerve and of the pneumogastric; the upper cervical nerves, however, are often involved. There are no morbid anatomical changes known. The most plausible theory of the disease is that it is a fulgurating neurosis, in which there are periodical discharges of nerve force, or nerve storms. The seat of the discharge is perhaps in the cerebral cortex, or possibly in the primary sensory centres, *i.e.*, the root ganglia of the fifth and vagus nerves. The disease is certainly not in the sympathetic system, as was once taught. The presence of excess of some endogenous poison as a factor in the disease may be regarded as most probable.

Diagnosis.—The diagnosis is based upon the hereditary history, the periodicity and seat of the attacks, the nausea, the complicating visual and other sensory symptoms. It should not be forgotten that the same patient may have migraine and other neuralgias or may have also an organic brain or renal disease.

Treatment—*Prophylaxis.*—Children of families in which this neurosis exists should be carefully watched during the ages between five and twenty. The eyes and nose should be examined. They should not be subjected to excessive mental or visual strain, and if attacks develop they should be promptly treated. The application of glasses should be considered, but not hastily adopted.

As regards constitutional treatment, the best measures for curing a case of migraine consist in correcting any visual or nasal defect, and prescribing such diet and exercise as secure health to the body and the nerves. Often a rest-cure or half-rest-cure is absolutely needed. The diet should be very simple and non-fermentative. It should be mainly of meat, fish, green vegetables and cooked fruits. The meats should be moderate in amount, and sometimes only fish and poultry should be allowed.

Of the drugs, alkaline tonics, given well diluted before meals, are the most trustworthy. Extract of cannabis indica may be given in large doses and for a long time (gr. $\frac{1}{3}$ to gr. i., ter in die). The salicylates are useful combined with an alkaline laxative like Rochelle salts, a dose being taken night and morning. Much stress is laid by some upon ocular muscular insufficiencies, and I believe that such conditions should be remedied, but place little confidence in them alone. On the other hand, the correction of small or large degrees of astigmatism and hypermetropia sometimes produces suprisingly good results. The reported cure of numerous cases of migraine by treatment of nasal hypertrophies and catarrh should not excite too much confidence in such measures. In fact, since migraine is a constitutional neurosis, one cannot expect permanent results from removing reflex irritants alone.

For *the relief of the attack* the measures recommended under headache are to be used. All drugs lose their effect after a time, and finally patients give up treatment or resort to codein or morphine. Chloral and a hot foot-bath break up attacks sometimes. Locally galvanic currents are sometimes helpful, and so are static sparks. Hot applications and pencilling with menthol give relief to some. Quiet and rest are spontaneously resorted to.

NEUROSES OF THE SENSORY BRANCH OF THE SEVENTH NERVE

As stated under the head of Facial Palsy, the seventh nerve has a sensory part, and its disorders cause otalgia of simple type, an "otalgic tic douloureux" and a herpetic otalgia, due to lesion of the geniculate ganglion. When both motor and sensory fibres are involved, there results the "Hunt syndrome" of facial palsy, otalgia and herpes of the ear,

NEUROSES OF THE ACOUSTIC NERVE

Anatomy.—The auditory or eighth cranial nerve has two different parts. One portion passes to the cochlea and utricle and saccules; it has to do with the sense of hearing; the other goes to the semicircular canals, and has to do with that sense by which we appreciate the position of our body and its relations to space. The eighth nerve is thus an auditory and a space-sense nerve.

The acoustic fibres enter the medulla by two roots, a lateral and a median. The lateral root has mainly auditory fibres. The space-sense fibres enter chiefly by the

median root. These roots are connected with three nuclei, viz.: (1) The chief nucleus, (2) Deiter's nucleus and (3) the accessory nucleus.

The lateral root is the one coming from the cochlea, and is, as stated, mainly a nerve of hearing.

The median root is connected chiefly with Deiter's nucleus (2) and through this with the longitudinal bundle and with the vestibulo-spinal tract and anterior horns of the cord. Fibres also go to the vermis.

The disease of the eighth or acoustic and space-sense nerve which are of special interest to the neurologists are auditory paralysis or nervous deafness, hyperacusis, tinnitus aurium, and auditory vertigo, or Ménière's disease. These diseases correspond with loss, excess and perversion or irritation of function.

NERVOUS DEAFNESS (ACOUSTIC PARALYSIS)

Nervous deafness may be due to lesions (1) of the two cortical centres of hearing, (2) of the acoustic nuclei, and (3) of the acoustic nerve itself or its end organ; (4) it may be psychogenous or hysterical. Practically, it is almost always the nerve and the internal ear which are affected.

Etiology.—1. Cortical nervous deafness has been known to occur in a few instances from lesions of both temporal lobes (Mills, Mott). The deafness of hysteria is of cortical (psychic) origin.

2. Deafness from lesion of the acoustic nucleus or nerve-root. Cerebrospinal meningitis in the young and syphilitic meningitis in the adult are the more frequent causes of this form. Fractures, tumors and hemorrhages may also be causes. When the auditory nucleus and nerve-root are affected by these diseases, its peripheral terminations in the labyrinth are also often involved, so that sharp distinction cannot always be drawn between this form and that due to labyrinthine disease.

3. *Deafness from Labyrinthine Disease.*—The causes are drugs, such as quinine and the salicylates; inflammations, including syphilitic exudates; injuries; hemorrhages; tumors; primary atrophy, which may be of family or hereditary type; and may occur in locomotor ataxia; mechanical causes, such as the constant noises and jarring to which locomotive engineers and boiler-makers are subject.

4. *Hysterical deafness* is caused by some morbid mental state or subconscious complex.

Symptoms of Nervous Deafness.—The dominant symptom is loss of hearing, but this may be accompanied by vertigo, tinnitus and even forced movements.

In hysterical or psychogenous deafness the loss of hearing is rarely complete, is usually unilateral, is more a bone conduction deafness and especially involves high and low notes. It is usually temporary. I have seen cases, however, where these characteristics were absent.

Deafness from involvement of the nucleus and nerve-root is usually accompanied by symptoms of a basilar meningitis or lesion of the pons

and medulla. Labyrinthine deafness is often associated with vertigo, tinnitus and forced movements, when it may become a symptom-complex known as "Ménière's disease."

Sudden total deafness is characteristic of hysteria and of syphilitic disease of the internal ear. In genuine nervous deafness, unless the deafness is absolute, hearing by bone conduction is lost or lessened, while aerial conduction is preserved. A tuning-fork vibrating on mastoid is not heard by the affected ear, though it is heard when held in the air close by this ear. (Rinne's test).

Normally a vibrating tuning fork with its base placed on the vertex of the skull is heard in both ears. If there is disease of the conducting apparatus it seems to the patient that he hears the fork in the ear of the affected side. This is called a "Positive Weber." In cases of disease of the auditory nerve, the sound of the fork will seem to be heard on the sound side. This is the "Negative Weber."

TINNITUS AURIUM. TINNITUS CEREBRI (NOISES IN THE EAR AND HEAD)

Subjective sounds resembling hissing, buzzing, humming, beating, muscial notes, etc., are classed together under the general head of tinnitus aurium. It is a very common symptom.

Etiology.—The disease attacks adults in middle and later life. Men and women are alike affected. Persons of neuropathic constitution and high blood pressure are more susceptible to the trouble. The arteriosclerosis of old age, cerebral congestion, sunstroke, tobacco and alcoholism predispose to it. It occurs often in involutional melancholia and in neurasthenia. Some local disease or congestion of the middle or internal ear is usually present.

Disease of the auditory nuclei and auditory tracts rarely, if ever, causes tinnitus; but chronic pachymeningitis, such as follows blows on the head, sunstroke, alcoholism, etc., may be attended by most annoying tinnitus, which is often not so much in the ears as in the head—a *tinnitus cerebri*. In old people with thickened arteries and imperfect brain nutrition a similar condition may occur.

Tinnitus accompanies confusional psychoses sometimes, and may be the source of aural hallucinations.

A kind of tinnitus may accompany migraine and it may take the form of an aura in epilepsy.

Despite this long list of causes, the chief factors may be summed up as neurasthenic states, local ear disease, humoral poisons and irritants and arteriosclerosis.

The *symptoms* are indicated by the name of the malady. They may come on suddenly, but usually develop slowly. Some deafness and

occasional vertigo are often present. The noise is located in one ear, as a rule. Sometimes it is said to be simply "in the head." The sounds are generally present all the time, giving the patient little rest and making life a burden. They vary greatly in character and intensity. These variations are indicated in the study of the diagnosis.

The Diagnosis.—The recognition of the symptom is easy. The principal thing is to discover its seat and cause.

The ear should, of course, be examined for external or middle-ear disease.

If the tinnitus is pulsating and synchronous with the heartbeats and stopped by carotid compression, it may be inferred that it is due to vasomotor paralysis or inflammatory congestion or aneurism.

If the sound is not in the ear, but in the head, and not associated with deafness or ear disease, the trouble is probably central, and most likely is of meningeal or arteriosclerotic origin.

Noises which are complex or take the form of musical sounds or words are probably central and illusional.

Constant rushing, knocking, pulsating noises are due to congestion, hemorrhage or inflammatory effusion in the labyrinth.

Moist sounds of a gurgling, bubbling, boiling, singing, whistling, shell-like roaring character indicate disease of the middle ear, with fluid exudation, catarrh of the Eustachian tube, or irritation of the external auditory canal or mastoid cells.

Dry roaring and ringing noises are due to non-suppurative catarrh of the middle ear, disease of the muscles or nervous supply of the tympanum.

The condition of the digestion should be inquired into and the existence of renal, arterial or central nervous disease investigated.

Treatment.—Ear disease must be treated if it is present. Hydrobromic acid and the other bromides, given in ordinary doses, are the surest palliating measures for this trouble. They may be combined with digitalis. Iodide of potassium and iodide of ethyl often are useful. Nitroglycerin is sometimes of value in patients with hard arteries. A combination of digitalis, bromide, and nitroglycerin has given very good results. Often general tonic measures are needed, and often also the patient must be taught to be patient with an ailment that cannot be cured.

VESTIBULAR NEUROSES (VERTIGO, DIZZINESS, GIDDINESS)

The vestibular nerve has to do with our sense of equilibrium, oculomotor control, orientation of the body and extremities. When it is disordered we get the symptoms of vertigo—staggering, nystagmus, nausea, and loss of power correctly to orient the extremities. To determine the condition of the vestibular nerve there are certain tests made.

The Barany Tests.—The vestibular nerve has its central connections with three ganglia in the medulla and from these ganglia it sends connecting fibres to the vermis of the cerebellum, to the posterior longitudinal bundle, probably fibres to the red nucleus and the cerebral cortex and via Deiter's nucleus to the motor cells of the spinal cord.

When this nerve is irritated, it produces the symptoms of dizziness, nystagmus, inco-ordination and disorientation.

The Barany test is one which enables one to determine whether the nerve is intact and can send its impulses through to its normal destination. The test in all its details is quite elaborate, but it is sufficient for ordinary routine neurological examination to use simply the cold water method.

The patient sits in a chair and the examiner injects half a dozen two-ounce syringefuls of cold water into the external meatus. The operator should be careful to have the water strike the side of the meatus and not be sent with any force against the membrane.

After this operation, if the vestibular reflex is normal the patient will feel dizzy and stagger, there will be a lateral nystagmus away from the ear which has been irritated and his finger will "pass the point," *e.g.*: if the patient is told to place the finger of his outstretched hand upon the finger of the examiner, shut his eyes, raise the hand to a vertical position and bring it down so as to touch the examiner's finger again, he will miss the finger and it will pass several inches to one side. If the hand of the patient is dropped to the side and the same test made, or if the arm is carried out horizontally at right angles to the examiner's finger, he will also "pass the point." If there is complete blocking of the nerve and the labyrinth is "dead" there will be no nystagmus, or vertigo or passing of the point. If there is no nystagmus it may be inferred that the connections of the nerve with the longitudinal bundle are affected.

The test is also made by using hot water, by the galvanic current and by rotating the patient while sitting on a stool.

Vertigo is a disturbance of consciousness characterized by apparent movements of external objects or of the person himself. If external objects whirl around, the vertigo is called *objective;* if the person himself seems to move, it is called *subjective.* Vertigo is almost always a symptom. In rare cases it appears to be *essential, i.e.*, due to a functional nerve discharge like the aura of epilepsy.

Vertigo is connected more or less with our space sensations, and hence it comes under disorders of the vestibular branch of the eighth cranial nerve and its central representations.

Etiology.—The causes of vertigo may be classed somewhat like those of headache, as follows:

(1) Hæmic, etc., anæmia, hyperæmia, gastro-intestinal and other endogenous toxins; toxæmia from tobacco and alcohol; (2) arterioscierosis, (3) vestibular nerve irritation; (4) neuroses: epilepsy, neurasthenia; (5) reflex: ocular, gastric; (6) organic brain disease; (7) mechanical causes, like electricity, swinging, etc.

Based mainly on the etiology, we have as a practical classification of ordinary cases of vertigo: autotoxic, arterio-sclerotic, vestibular, ocular and neuropathic forms. The various causes of vertigo act by irritating the space-sense nerve and thus disturbing our sense of relation to external objects, and by irritating cortical centres. Vertigo is a painful response of the vestibular nerve as neuralgia is a painful response of the fifth nerve. Vertigo may be due to organic lesions of the cerebellum and its peduncles; it is then associated with forced movements of the body.

Symptoms.—Vertigo comes on suddenly, and lasts, as a rule, for but a moment. The floor rises and sinks, or objects whirl around (objective vertigo), or the patient seems whirling around or falling. The ideas are confused; there are a sense of alarm and a feeling of faintness. The patient totters, sometimes falls; there may be nausea or vomiting. In some forms there is momentary loss of consciousness or syncope. Vertigo usually comes on in short attacks, but in toxæmic states, as in alcoholism or nicotinism, it is almost constant while the poison is in the system. Vertigo may become chronic or nearly so; and if severe it forms what is called the *status vertiginosus* (Mitchell). Vertigo is increased by rising or sudden movements and lessened by lying down.

Symptoms of Special Forms —*Auditory Vertigo* (*Ménière's Disease*).—A large proportion of severe periodical vertigoes are due to disease or irritation of the eighth nerve and its centres. The common cause is local disease of the labyrinth. When this produces severe attacks of vertigo with nausea and perhaps syncope, it is called "Ménière's disease." The name is often applied to any form of auditory vertigo. Ménière's type is always due to organic disease of the labyrinth. It is usually accompanied by progressive deafness, and sometimes by tinnitus and forced movements or even utter inability to walk steadily. When the deafness is complete the vertigo ceases, because the nerve end-organ is destroyed. Mild forms of auditory vertigo present nothing unusual except those due to involvement of the nerve of hearing.

Bilious and Stomachal Vertigo.—In condition of dyspepsia, constipation and hepatic torpor, the loaded and disordered stomach and bowel suddenly discharge into the blood irritant substances which pass to the brain and by direct action on the nervous centres cause vertigo. This is probably the explanation of the vertigo of biliousness and constipation. It is a paroxysmal vertigo, noted most in the morning, not very severe,

and often accompanied by nausea. Stomachal vertigo is more acute, severe and may be accompanied with loss of consciousness.

Neuropathic Vertigo.—The symptoms of epileptic vertigo will be described under that head.

Neurasthenic vertigo is a not uncommon symptom. The attacks are short, generally subjective, not severe or accompanied by nausea or syncope, but they often cause much alarm. Underlying them are exhausted and irritable nerve-centres, with ocular, gastric and humoral irritations or a weak heart.

A neuropathic vertigo occurs sometimes in the form of attacks almost exactly resembling seasickness. There are intense vertigo, nausea, and faintness lasting for hours, coming on suddenly without known cause except overwork or excitement. The attacks occur in neurotic subjects and are analogous to the other nervous crises. It is a periodical neurosis of the space-sense nerve and resembles migraine.

A form of vertigo which is *psychical* in character occurs in neurasthenics. It consists in a sudden sensation of insecurity, an apprehension of falling, of an approaching loss of consciousness. There is no true vertigo, either subjective or objective, and the patients really never stagger or fall. It is a psychosis rather than a nervous condition.

In some nervous subjects there occurs a sudden giving way of the legs. There is no conscious vertigo, yet such probably exists. The symptom is noted in exophthalmic goitre, paralysis agitans and epilepsy.

Ocular vertigo is a rare symptom, but is, when present, chronic and annoying. It is caused by refractive errors and unequal action of the ocular muscles.

The *mechanical vertigoes*, such as seasickness, car sickness, etc., are produced by swinging or whirling, the movements of the ship, steam car and elevators. Railway mail clerks, elevator boys, often suffer from chronic disturbances of a vertiginous character. Ocular and auditory nerve sensations enter mainly into the causation of the troubles.

Arteriosclerotic Vertigo, Senile Vertigo.—This occurs in persons who have arteriosclerotic changes in the brain or labyrinth vessels, either from disease or senility. The symptom is caused by impaired brain nutrition with consequent anæmia. Senile vertigo may also be due to a weak and fatty heart.

Diagnosis.—In investigating vertigo the physician should find (1) whether it is subjective or objective; (2) paroxysmal or chronic; (3) accompanied by ear symptoms, nausea, tinnitus, and loss of consciousness. He should then direct himself to finding the special cause and seat, remembering that the vestibular, gastric, toxic, and neurasthenic are the common forms. In elderly persons the arteries should be carefully

examined. In young persons the possibility of epilepsy must be remembered.

The prognosis depends upon the cause. Epileptic vertigo and vertigo from organic disease are most serious. Ménière's vertigo usually ceases when complete deafness occurs. The other forms of vertigo are ordinarily susceptible of relief.

Treatment.—The attack is treated by rest in the horizontal position and the administration of a volatile stimulant. The disorder must then be treated in accordance with the cause.

In Ménière's vertigo the use of quinine by Charcot's method is said to be useful. Quinine is given in doses which are gradually increased until slight cinchonism results; then the drug is stopped. Mitchell advises the addition of hydrobromic acid; Gowers advises the use of salicylate of sodium in five-grain doses instead of quinine. Hirt recommends ten drops of a 2 per cent. solution of pilocarpin injected hypodermically every other day. A better method is to give a sweating dose of pilocarpin muriate, gr. $\frac{1}{4}$ to $\frac{1}{10}$, with a tumbler of hot water early in the morning before the patient rises.

Neurasthenic vertigo is cured by rest and attention to diet, laxatives and mineral acids being used. Hydrobromic acid with pepsin and glycerin are often very helpful here. Gastric vertigo is to be treated with saline laxatives and simple bitters before meals.

In the vertigo of "biliousness" and constipation there is often a neurasthenic element, and a similar attention to diet and to the digestive organs is indicated. In arteriosclerotic and senile vertigo small doses of nitroglycerin and iodide of potassium, with or without digitalis, should be given. Rest and warmth of the extremities are indicated. In all forms of vertigo bromide of potassium is helpful and will relieve the symptoms for a time. It is the best symptomatic remedy.

There are two peculiar forms of disease to which the name vertigo has been attached which may be described here.

Laryngeal Syncope (*Laryngeal Vertigo, L. Epilepsy*).—This is a rare form of disorder characterized by attacks of paræsthesia of the throat, with coughing, followed by sudden syncope, and sometimes by slight convulsive movements.

At the onset of the attacks a burning or tickling sensation is felt in the larynx or trachea; there is a spasmodic cough, perhaps some asthmatic or dyspnœic symptoms, when the patient suddenly falls unconscious for a short time. The attacks may occur daily or only once in a few weeks. The disease is an epileptic or tabetic phenomenon, or due to local disease.

The treatment should be directed to relieving the cause.

Paralyzing Vertigo (*Gerlier's Disease*).—This is a disease occurring only on the farms in southern France and Switzerland.

The symptoms consist of sudden attacks of ptosis, vertigo, paresis of arms and legs and cervico-occipital pain. The disease is most prevalent in the summer time.

It attacks chiefly males. Single attacks last not over ten minutes, but may occur frequently.

The cause is supposed to be a special microbe developed in the stables during the heat of summer.

We are not aware of its occurrence in America, though Seguin called attention to the close similarity of the symptoms to those of poisoning by conium maculatum.

SENSORY NEUROSES OF THE GLOSSOPHARYNGEAL NERVE

The anatomy of this mixed nerve is described under the head of motor neuroses. The sensory fibres may be affected in hysteria, causing the symptom called globus, and also the pharyngeal anæsthesia found in the same disease.

The special fibres of taste may be affected, causing ageusia or loss of taste.

Ageusia (*loss of the sense of taste*) is an affection in which the power to discriminate the tastes of bitter, sweet, salt, acid and alkaline substances is lost.

Etiology.—Hysteria may cause a hemiageusia. Lesions of the corda tympani, of the third root of the trigeminus or of the glossopharyngeal nerve may cause more or less ageusia. Catarrhal diseases of the mucous membrane of the mouth and nose are frequent causes. It is not caused by cortical brain disease so far as known. Some ageusia is present in the imbecile, and the sense of taste is less keen in the lowly organized and criminal classes.

Symptoms.—The symptoms are subjective and may not be noticed at first by the patient. In hemiageusia from facial palsy and in hysteria it has to be looked for, as the patient does not complain. The tests are made with solutions of salt, sugar, vinegar, and quinine. A single solution of sugar usually answers. But the different parts of the tongue differ in sensibility to different substances. Care must be taken to exclude the nose as a factor in taste.

Disease of the trigeminus and facial usually causes ageusia on the anterior two-thirds of the tongue, with loss of taste, especially for sour and bitter substances. Sometimes, however, disease of the trigeminus or disease of the tympanum involving the tympanic plexus and chorda tympani causes ageusia of the whole tongue on the affected side.

Ageusia from disease of the glossopharyngeal alone is very rare. there is loss of taste on the posterior third of the tongue, soft palate and pillars of the fauces, to sweets and acids. A few cases have been reported in which paralysis of the glossopharyngeal caused complete ageusia on the affected side. It must be inferred, therefore, that taste fibres run sometimes wholly in the fifth, more rarely wholly in the ninth nerves, and usually in both.

The *treatment* depends on the cause. Locally, cleansing and stimulating mouth-washes and electricity may be used.

Parageusia, or perversions and imperfections in the taste sense, are very frequent. They are generally due to irritation of the taste nerves from catarrhal inflammation of the stomach or mouth. They also occur in hysteria and in syphilis.

SENSORY NEUROSES OF THE UPPER CERVICAL NERVES

The mass of muscles at the back of the neck is made up of nine individual muscles on each side. They are innervated by the posterior branches of the upper six cervical nerves and the spinal accessory.

Sensation is supplied to this muscular mass and to the back of the head and neck by the anterior branches of the second, third and fourth cervical and the posterior branches of the second cervical. The cutaneous distribution of these nerves is shown in the diagrams. (Figs. 27 and 28.)

From a study of the innervation of the skin and muscles of the neck, it seems that most neck pains and paræsthesiæ are placed in the areas supplied by the occipitalis major and minor nerves, branches of the second and third cervical nerves. (Fig. 75.)

Cervico-occipital Pains.—There are practically only a few neuralgias which may be considered to have a definite clinical course and to be entitled to the name of special diseases. The chief of these are trigeminal neuralgia, neuralgia of the brachial plexus and the neuralgia of the sciatic plexus. The other forms of neuralgia are nearly always symptomatic or reflex pains or are distinct manifestations of a neuritis or some injury or irritation of the nerves. Pains in the neck, therefore, are very rarely to be classed under the head of "cervical" neuralgia or "cervico-occipital" neuralgia. Nevertheless, there is no region of the body in which pain is more frequently complained of than in the back of the neck and head, unless we except the trigeminal pains. The excessive amount of pain experienced in the neck is due to several causes. The muscles of the neck are continuously used, perhaps more than almost any other muscles, except those of the eyes. The supporting of the head in the erect posture and the movements of it which accompany the use of vision make the functions of the upper four cervical nerves an almost continuous performance.

Whenever a group of muscles is thus used to such a continual extent in function two morbid phenomena are apt to occur. One is an exhaustion of these muscles, with consequent pain from their fatigue; the other is the development in the muscular bodies or their connecting tissues and nerves of rheumatic and vascular disturbances—so that

pains in the neck are, as a rule, either exhaustion pains or rheumatic pains.

There is, however, another source of pathological disturbance. Whenever a serous membrane is irritated, the muscular bodies which surround it and by their movements and arrangements support it, experience reflex disturbances. This we see exemplified by the pain in the side when the pleura is involved and the pain about the joints when they are inflamed. So whenever the serous membranes surrounding the brain become congested or irritated, there results a reflex disturbance upon the muscular support of the skull in which the brain is enclosed.

Whenever, then, by a persistent over-use of the brain, there occur vascular changes of fatigue, the neck muscles show it in a reflex irritability and various accompanying sensory disturbances. So in the beginning of meningitis we find the pain in the neck one of the earliest of the storm signals sent out.

Etiology.—Pains in the back of the head and neck occur in migraine, spinal irritation and neurasthenia, as a result of eye-strain, as a true neuralgia and as a symptom of brain tumor, meningitis and from a rheumatic inflammation of the neck muscles and nerve. Trauma, spinal tumors, local disease of the vertebræ and herpes zoster are causes of cervical pains. Cervical neuralgia may occur in tabes.

Symptoms.—Migrainous neck pain is described elsewhere. It is a one-sided trouble and periodical in type. In spinal irritation the pain is central or, perhaps, shifting; it is especially characterized by a sharp, boring pain just below the occiput. With it there may be evidences of cerebral congestion with vertigo and faintness, and even vomiting. The boring pain is almost pathognomonic of spinal irritation. In neurasthenia which is often associated with an irritable spine, the pain is more of a diffuse, aching character.

In typical neuralgia the pain is usually unilateral, paroxysmal and sharp, sometimes reaching the intensity of a tic douloureux. There are tender points over the exit of the nerves. The disease lasts for five or six weeks. The pains may alternate with or take the place of a trigeminal neuralgia. The nerves involved are the great and small occipital from the second pair and a branch from the third pair. Sometimes there is a true neuritis due to herpes zoster and then cutaneous anæsthesia is present.

Treatment.—Symptomatic constitutional treatment is indicated, viz., rest, correction of eye-strain, tonics, and when indicated the use of antirheumatics, such as the salicylates. In women pelvic troubles should be looked for. Locally, counterirritants, cupping and leeching are useful; mustard and capsicum pastes are often a great relief. The ice bag also

is of service. Resection of the occipital nerves has cured one obstinate case.

SENSORY NEUROSES OF THE LOWER CERVICAL AND BRACHIAL NERVES

Brachial Neuralgia and Neuritis.—Definition and Frequency.—Neuralgia of the brachial plexus is a disease characterized by severe pains centering in the upper arm but usually involving the whole upper extremity and shoulder; it runs a course of several months and is due to irritation with congestion of the trunks of the brachial plexus and its roots of origin. This irritation does not usually reach the degree of neuritis, and the disease does not show the paralysis and atrophy, anæsthesia and the vasomotor symptoms of neuritis, as a rule. This form of neuralgia, even excluding the cases of traumatic neuritis, is now by no means a rare disease. Among the neuralgias brought to the attention of the neurologist it ranks second, coming after the trifacial neuralgias and followed in frequency by the sciaticas.

Total cases of arm pains	79
Total brachial neuralgias	41
Arm pains associated with distinct neuritis	15
Occupation neuroses with dominant arm pains	16
Hysterical arm pains	4
Palmar, digital and hand pains due to local or reflex causes	6

Etiology.—Brachial neuralgia occurs oftenest in persons in middle life, ranging from thirty-five to fifty. When it occurs in young people it is (like the other neuralgias) more often of the hysterical type, or at least associated with those persons who are neurotic and over-sensitive. Besides having an hereditary neurotic history as the underlying tendency, there is almost always a history of severe emotional strain—loss of friends, sickness in the family or worry over complications of domestic life. Sometimes, in addition, there is some physical injury or some acute sickness. In two cases I have seen it follow profuse hemorrhages. In some cases it is due to inordinate use of the arms in simple manual work, like embroidery or knitting; but this seems to me rather unusual. A mild form of brachial neuralgia occurs in women as the result of carrying a heavy skirt, and it is not unlikely that this kind of work (skirt carrying) and the use of the arms in dressing the hair bring on neuralgia in predisposed cases.

Brachial neuralgias are seen relatively oftener in the better classes than in dispensary or hospital patients. They occur oftener in women than in men in the proportion of about three to one. This is contrary to most statistics, because, as I believe, these statistics include many frankly traumatic and surgical cases. There is often a predisposing

neurotic or rheumatic taint, and sometimes perhaps an autotoxæmia. Brachial neuralgia occurs in tabes dorsalis and as a symptom in other organic diseases. In fine, the etiology of brachial neuralgia has always one or more of three factors: neurotic constitution, exhaustion from occupation or trauma and some endogenous toxæmic irritation.

Pathology.—The common form of arm pain is usually designated as a neuralgia, though there is probably some low grade of inflammatory process in the sheaths of the nerves. If it is desired to use the term "neuritis," I do not know that serious objection could be made. So far as I am aware, no post-mortem examinations have been made of the nerves of persons suffering from this condition, except of certain acute hemorrhagic cases. If we may infer concerning it by analogy, from conditions seen in sciatica, the existence of perineuritic irritation and perhaps slight exudate, is probable. Probably sometimes the inflammation is not so much in the nerve-sheaths as in the muscular and connective tissues about it, and that there is a myositis as well as secondary neuritis. In using the word "brachial neuralgia" I include both the neuralgias, with no objective symptoms, and the distinctly neuritic cases, of non-surgical origin.

Symptoms.—The onset is rather sudden, but there may be severe preliminary aching in the arm for a few days, and sometimes there are short preliminary or abortive attacks. Usually, however, the patient wakes at night or notices in the morning a distinct and severe pain involving the upper arm and shoulder or perhaps the whole arm. The pain usually is most acute, however, on the inner and front side of the arm and in the back between the shoulders. It is very intense and runs down the forearm and into the fingers, involving sometimes the whole hand, but usually only the first, second and third fingers. It is not a darting, shooting pain, but seems to be one that is general and diffuse, involving for a time the whole arm with a pain of paralyzing intensity. The pains exacerbate, coming on usually at night more severely, or more in the morning, and always more after exertion. Movement makes the suffering worse; but the arm can be handled gently without much pain. Some suffering is present nearly all the time, often robbing the patient of sleep; but it may let up for a few hours during the day and then comes back again with paroxysms of great intensity lasting for one or two hours.

Many patients try at first to work off the pain by exercise, thinking it a form of rheumatism, or they get a masseur who rubs the arm, and, as a rule, makes it worse. There are, however, at times, attacks that are short and abortive. I have known some to last three or four days, and in these instances the massage or the doctor get the credit for a therapeutic triumph entirely undeserved. Usually the pains progress and

continue for two or three weeks until the trouble is pretty firmly established, continuing then for two or three months.

When the pain first comes on there are no objective changes in the arm. It looks normal and is simply tender and sore to the touch. Later the arm becomes a little swollen and the fingers somewhat edematous, and there is slight flabbiness of the tissues, showing vasomotor change.

In the typical neuralgias there is rarely much more than this. If it is a genuine neuritis, atrophies and paralyses gradually develop. In brachial neuralgias proper the changes in the arm are only slight—the grip of the hand becomes weak, the elbow-jerk is a little exaggerated at first, muscular irritability is increased and then diminished, and there is no anæsthesia.

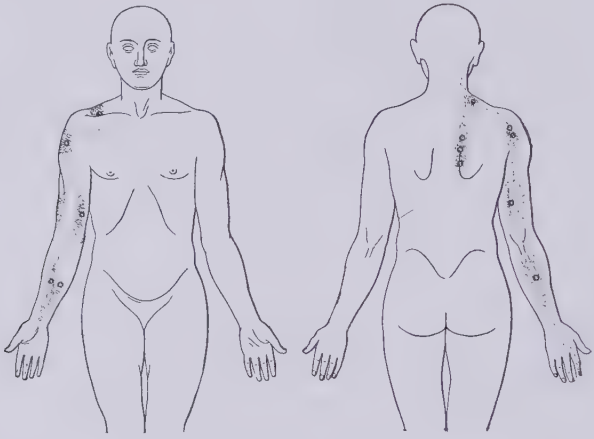


Fig. 78.—The pains and painful points of brachialgia.

Examination shows points of tenderness along the course of the nerve on the inner side of the arm at the elbow and over the deltoid. There is also a point of tenderness between the scapulæ about at the level of the second or third dorsal vertebra. This is very painful and is characteristic. Stretching out the arm and drawing upon the brachial plexus brings out pain, just as it does in the extension of the leg in sciatica. Later there is also weakness of response to the elbow-jerk, just as in the tendo achillis in the later stages of sciatica. The tender points that one finds in brachial neuralgia are by no means always identical with the tender points of Valleix. They vary somewhat and only correspond in a general way to the distribution of the cutaneous nerves (Fig. 78).

It will be found, as we note the neuralgias from the back of the neck down to the hand, that pains in these different segments are associated with painful points along the course of the spine. For example: In occipital neuralgia there will be a well-marked tender point close up to

the base of the skull over the second or third cervical spines. With a cervicobrachial neuralgia the point moves down to near the first or second dorsal. With a brachial neuralgia the spinal tender point moves to the neighborhood of the third or fourth dorsal. These spinal tender points are quite characteristic of neuralgias of the upper cervical plexus, and the collaterals of the brachial plexus.

Diagnosis.—In the chronic and less severe types of arm pain one may be easily misled in diagnosis. The first and obvious name given to an arm pain is "rheumatism." The patient is always treated first for this.

It is safe to say that arm pains are never purely rheumatic, unless there is found some objective evidence of myositis, arthritis, or periarthritis. The most difficult thing is to distinguish between real neuritis of high grade and neuralgia. Neuritis has become a popular diagnosis nowadays, and patients talk very glibly about the term. But a neuritis cannot be recognized except by some objective signs. There must be pain and also tenderness along the nerve; there must be definite areas of hyperæsthesia or anæsthesia, muscular weakness, and perhaps atrophy and electrical changes. There must be some of these symptoms in an inflammatory process that last three to six months.

In about half of the cases no such objective symptoms are present. The condition is not one of actual inflammation, but of exhaustion, and in the majority of cases brachial neuralgia is an exhaustion neurosis with some rheumatic or metabolic irritation of the nerves in addition.

When we find a herpes along with arm pain we know that we have an inflammation of the spinal ganglion and secondary neuritis and that the process is due to an infection.

Treatment.—The treatment of brachial neuralgia is fundamentally that of rest. This must be secured by a sling, and, if necessary, by splints. In a good proportion of cases some relief is gotten by the salicylates, especially aspirin, but they must be given in very large doses, just as for acute rheumatism.

No massage nor other mechanical treatment should be given at first. But hot air, baking, hot pads, hot applications of *mud* or flannel should be used. The patient must understand that, as a rule, it often takes ten or twelve treatments to accomplish results.

After a course of salicylates, tonics can be given. I do not know any drug that is of any special value unless it is strychnine, in massive doses, but of its utility I am sure in chronic cases.

Arm pains, which once were due to neuritic conditions, sometimes become habit pains; the patient has a "constitutional armache," just as some have constitutional headache. The best and only treatment for these patients is to ignore the trouble absolutely. When the pains have become chronic, *i.e.*, after four to six weeks, massage and electricity

do some good. The patient gets well anyway in three to six months as a rule.

The disease may recur, but this is rather rare, and only in about 10 per cent. of my cases.

It is rarely double, and when so is invariably an exhaustion or psychogenous neuralgia, if we can exclude organic disease.

Pains in the Forearm and Fingers.—There are rarely true neuralgic or neuritic pains confined to the *forearm* alone. Here we have only the ulnar pains following the course of the ulnar nerve, and very characteristic radial pains following the back of the arm to the fingers, and least frequently median nerve pains. Sometimes the whole hand is involved in neuralgic pain, but this is usually hysterical. The hand and

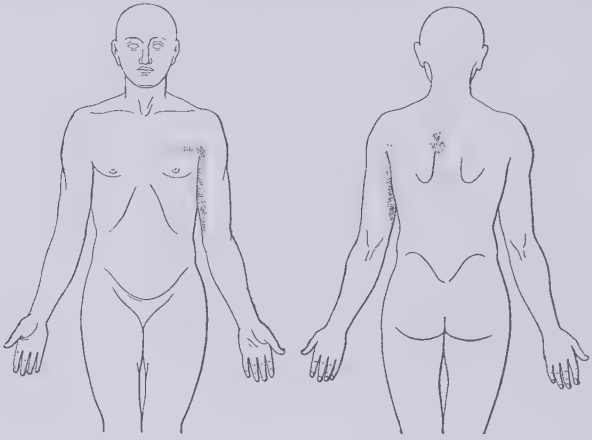


Fig. 79.—The location of reflex heart pains.

fingers have paræsthesias and local pains due to central or local disease, but no neuralgia except in tabes dorsalis.

Heart Arm-pains.—Cardiac arm pains are not very prominent factors in the ordinary heart disease of the hospitals, and do not appear so dominant among the laboring classes. Almost always in persons who have a cardiac disease and who have pains in the arm there is a decidedly neurotic element. This means, that the nervous system being more sensitive and unstable, the irritating impulses from the myocardium radiate more freely and arouse disturbances in consciousness more easily. These cardiac arm pains, are generally associated with some precordial pain, not infrequently with some pain near the scapula, and also pains on the inner side and middle of the upper arm and middle of the forearm. They rarely extend into the fingers. Sometimes there is distinctly a pain in the right arm instead of the left. The accompanying diagram shows the usual localization of these pains (Fig. 79). There

is generally a feeling that the pain comes from the left side and runs from there into the arm. The cause of this reflex disturbance is generally attributed to the fact that the third or lower cardiac ganglion of the cervical sympathetic sends fibres which communicate with the first dorsal nerve, which nerve supplies the inner side of the arm (Fig. 80).

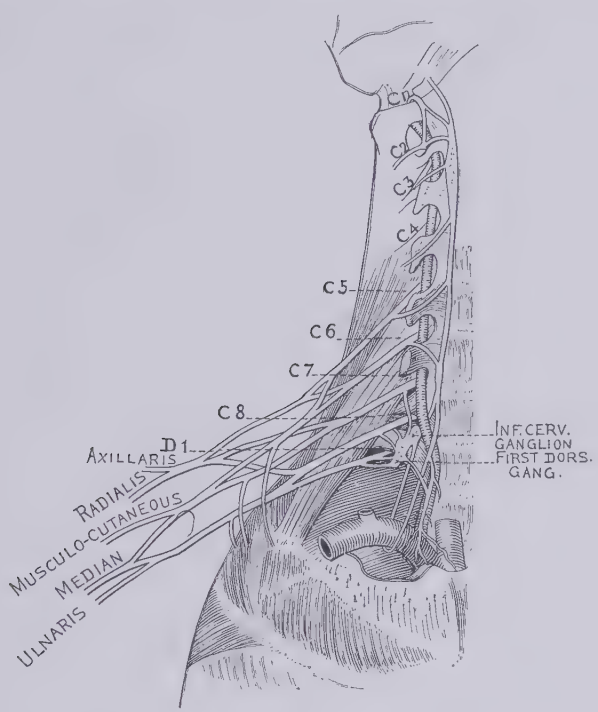


FIG. 80.—Showing the relation of the third cervical and first thoracic ganglion (blended together) with the brachial plexus.

THE SENSORY NEUROSES OF THE INTERCOSTAL NERVES

These are chiefly intercostal neuralgia, symptomatic side pains, and herpes zoster. Paralyses and anæsthesias occur in connection with vertebral and spinal-cord diseases.

Intercostal Neuralgia.—Intercostal neuralgias are quite rare. They occur in chronic lead-poisoning, in the neuralgic form of tabes, following herpes zoster, and in hysteria. Neuralgic pains in the side due to some organic disease irritating the intercostal nerves are, however, common. They occur in vertebral and spinal cord disease (tumors and syphilis) in local disease irritating the nerve, fracture of the ribs, pleurisy, tumors, aneurism, myositic and arthritic processes, angina

pectoris, and trauma of the nerves. Reflex pains from disease of the viscera are common (see Fig. 68).

Symptoms.—The pain in typical cases is sharp and stabbing, but not much increased by respiratory movements. There are tender points at the seat of pain, which is usually greatest over the side at the exit of the lateral nerve branches. Often a tender point is felt over the exit of the dorsal or of the anterior branch. It is rare to find all three points.

The sixth to tenth nerves are those oftenest involved. The left side is more susceptible than the right.

Probably one-half the pains in the side are myalgic in nature, and should be classed as such. These pains can be distinguished by the history of their origin and of rheumatic influences, by their diffuseness and dullness, by the great tenderness on pressure, and the pain produced on taking a deep breath. There is another considerable proportion of cases in which the pains are mainly neuritic, but yet there are some eivdences of muscular complications. Some of these pains are reflex.

In the third class of cases there is the pure stabbing neuralgia. The diagnosis is based on the exclusion of pleurisy, visceral disease, tabes, rheumatic causes, by the character of the pain and the presence of tender points.

Treatment.—In all cases the heart, pleura, stomach, and pelvic organs must be examined, and any disorder relieved. If there is a rheumatic and muscular element, salicylates or the analgesics should be given. Apply heat and secure rest by adhesive straps.

Mammary neuralgia (*mastodynia*) is a form of intercostal neuralgia involving the anterior and lateral branches of the three or four upper dorsal nerves.

Etiology.—It may be caused by local disorders, or it may be a functional or a traumatic neuralgia. The causes in the latter class are pendent breasts, pressure from badly-fitting corsets and injury. Mammary neuralgia also occurs in hysterical women and young girls sexually precocious; it may occur in pregnancy and during lactation. Many mammary pains are due simply to local disorder of the gland.

Symptoms.—Mammary neuralgia is unilateral, often very severe, and if it occurs in middle life is liable to cause much mental depression from fear of cancer.

The *treatment* depends upon the cause. It requires general tonic measures and attention to reflex agencies and to the proper support and protection of the gland.

Herpes Zoster (*Shingles*).—This is an acute dermatitis, secondary to a ganglionitis. It may attack any of the cerebrospinal ganglia.

Etiology.—Its predisposing causes are wounds, drug habits, rheumatic, gouty and syphilitic poisons and emotional influences. The active cause is in most cases an infection, and the disease sometimes is almost epidemic. The trouble is not infrequent in tabes dorsalis. The inflammation affects not only the nerves, but the spinal ganglia, and especially the latter, so that the condition has been called a posterior poliomyelitis (Head). Examination of the cerebrospinal fluid often show a lymphocytosis.

Symptoms.—It begins gradually with the development of pain and a herpetic eruption upon one side of the trunk. It generally involves the lower dorsal nerves. The eruption follows the course of the nerve, rarely extending to the opposite side. The pain gradually subsides, and the disease itself runs its course in one or two weeks. Sometimes it leaves the patient with a chronic neuralgia lasting for months.

Treatment.—Local anodynes and protective ointments should be applied (1 per cent. mixture of cocaine in lanolin). Antirheumatics and analgesics are to be administered internally. The free use of the analgesic coal-tar products appears to lessen the severity and length of the disease. In chronic cases very large doses of strychnine are indicated.

SENSORY NEUROSES OF THE LUMBAR NERVES

Painful conditions center about the lumbar region and hips more than in any other region except the head. Most of these pains are myalgic, and arthritic, or are reflex from pathological conditions in the pelvis and lower abdominal cavity. Such pains are diffuse and generally bilateral.

These various pains are grouped clinically under the head of:

1. Lumbago.
2. Spinal irritation (neuro-myalgia).
3. Arthritis of various types and joints, from gonococcic and other infections.
4. Reflex pains.

This leaves few disorders of the lumbar nerves that are strictly neural in origin. They are lumbo-abdominal neuralgia, meralgia and anterior crural neuralgia.

Lumbo-abdominal Neuralgia.—The upper two lumbar nerves are almost entirely sensory. When they are the seat of a neuralgia, there is sharp pain in the groin, back and buttocks, extending over the hypogastrium or genitals on one side. The pain in the back, however, is often bilateral. Painful points may be found after a time, as in intercostal neuralgia. Sometimes the pain is located in the side of the penis (penile neuralgia).

Crural neuralgia of the long lumbar branches is called femoral or

crural. When these nerves are subject to a lesser irritation, causing sensations of numbness and pricking along the thigh, the condition is called *meralgia*. In true neuralgia, the patient complains of pain in the front of the knee and the anterior and outer parts of the thigh, but has no pain posteriorly and none below the knee. The internal branches of the anterior crural nerve do not seem to be affected, while the middle and external cutaneous branches and the genito-crural nerve are involved.

Reflex Pains.—Disease of the hip or of the sacro-iliac joint or vertebræ may cause a reflex pain in the obturator nerve, localized especially in the

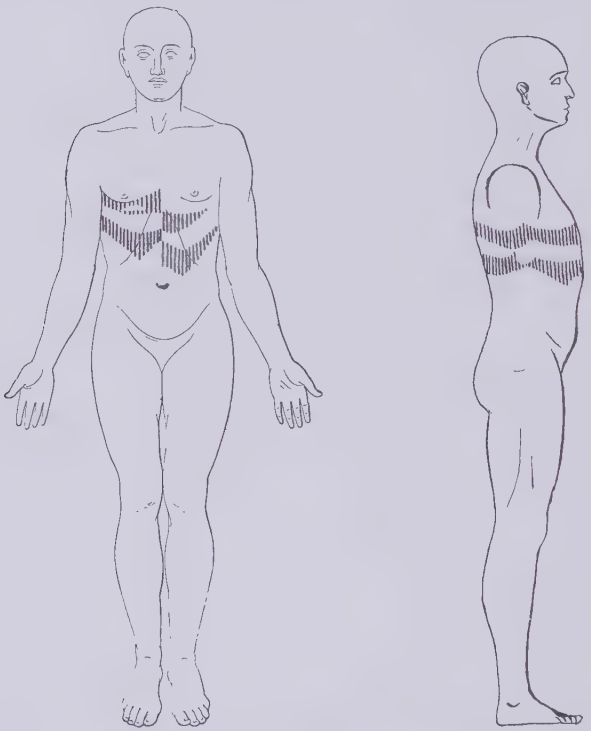


FIG. 81.—Areas of cutaneous hyperæsthesia in disease of the stomach. Sixth, seventh, eighth and ninth dorsal nerve-segments. (*Head—Fraenkel.*)

knee and back of this joint. The fœtal head sometimes compresses these branches, causing a symptomatic neuralgia. Disorders of the womb and its adnexæ are especially liable to cause reflex pain in the lumbar nerves. Lesions of the appendix, kidney, ureters also reflect pains to the lumbar nerves. Diseases of the external genitals and bladder more often reflect pains upon the sacral nerves. In renal colic pains are felt in the ilio-inguinal and hypogastric nerves. Local disease of the psoas muscle or

iliacus, in the neighborhood of Poupart's ligament, causes pains in the lumbar nerves. (See Head Zones, Figs. 81, 82, 83, 84, 85).

Pains over the sacro-iliac synchondrosis occur in rheumatic inflammation of these joints. Goldthwaite has shown that in certain injuries there may be a slight dislocation and sprain of these joints. In these cases if the patient lie on his back and the leg lifted in the extended position, great local pain is elicited and the patient cannot voluntarily raise the extended leg.

Diagnosis.—Lumbar neuralgia is distinguished from lumbago by the unilateral position, the distribution and paroxysmal character of the pain and lack of severe suffering on motion and pressure; the tender points and the absence of any organic disease. Lumbago comes on suddenly, with a history of exposure or strain, is bilateral and confined to a single group of muscles, which are tender on deep pressure. In lumbar strain the onset is also sudden, with a history of injury, great local tenderness and evidences of trauma. Most chronic lumbar pains are reflex or symptomatic and the main object of diagnosis is to find the cause.

The *treatment* is the removal of the cause. The frequent presence of pelvic disease, of sacro-iliac joint disease, and of strain and a rheumatic history must be borne in mind.

Head's Zones.—In cases of acute or subacute inflammation of the viscera, areas of skin hyperæsthesia usually appear in certain definite segments corresponding to the organs diseased. The accompanying figures are furnished me by Dr. Jos. Fraenkel from cases observed by him. The testing for these hyperæsthetic areas has sometimes diagnostic value.

Sciatica (*Neuralgia of the Sciatic Nerve, Sciatic Neuritis*).—This is a form of neuralgia occurring in middle life and characterized by intense pain in the course of the sciatic nerve. A large proportion of the cases is due to neuritis.

Etiology.—The disease occurs three times as often in men as in women, and is the only neuralgia of which this can be said. Most cases in this country occur between the ages of forty and fifty; next between thirty and forty.

The gouty and arthritic diathesis, and occupations which lead to exposure and strain, predispose to the disease. It is not rare, therefore, among laboring men. In younger persons a neurotic constitution predisposes to the disease, and in this class the trouble is more truly of a reflex and neuralgic character and less of a neuritis.

Most cases occur in the autumn and winter. The exciting causes are constipation, pressure from hard seats, exposure, muscular strain from heavy work and pelvic disorders. Symptomatic sciatica may be

caused by the pressure of pelvic tumors, injury to the nerves, vertebral and spinal disease; sciatica occurs in paralysis agitans and in diabetes and in phthisis. In elderly persons of a rheumatic constitution inflammatory processes about the hip-joint complicate or cause the neuralgia.

Symptoms.—The disease begins rather suddenly. Pain is felt in the back of the thigh, running down the leg in the course of the nerve. Generally it is most marked in the thigh, extending up often into the lumbar region. Sometimes the disease begin like a lumbago; more rarely

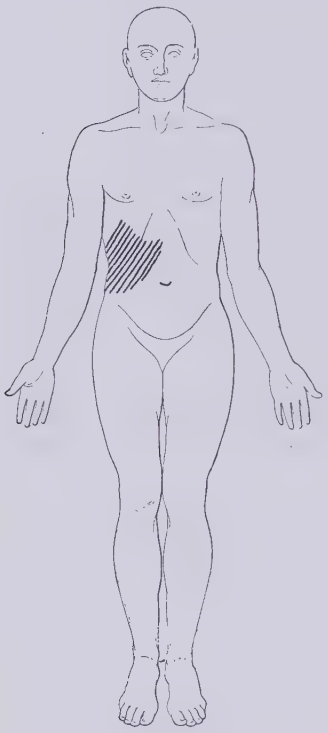


Fig. 82.

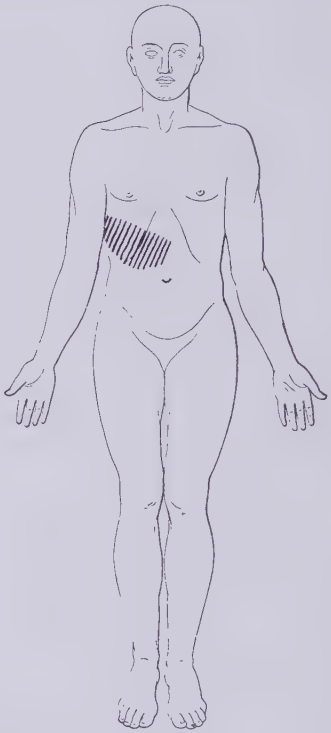


Fig. 83.

Fig. 82.—Area of cutaneous hyperæsthesia in a case of cholelithiasis and cholecystitis. (*J. Fraenkel.*)

Fig. 83.—Area of cutaneous hyperæsthesia in a case of cholecystitis. (*J. Fraenkel.*)

pain is first felt in the calf or foot. The pain is increased by motion, and the patient holds himself in a constrained position. The pelvis is tilted up toward the sound side and the trunk inclined over to the diseased side. After a time this leads in some cases to a characteristic deformity (sciatic scoliosis) in which the convexity of the curve of the vertebral spines is directed toward the diseased side. The pain is almost continuous, with paroxysms of great severity, which often occur at night. During these

paroxysms the pain is sharp, burning and lancinating. In the interval it is dull. Besides the pains the patient suffers from feelings of numbness, tingling, and a sense of coldness and weight in the affected limb. There are almost always tender points over the course of the nerve. These may be found at the sciatic notch, at the middle of the hip, behind the knee, just below the head of the fibula in the middle of the calf, behind the external malleolus and on the back of the foot (Fig. 86).

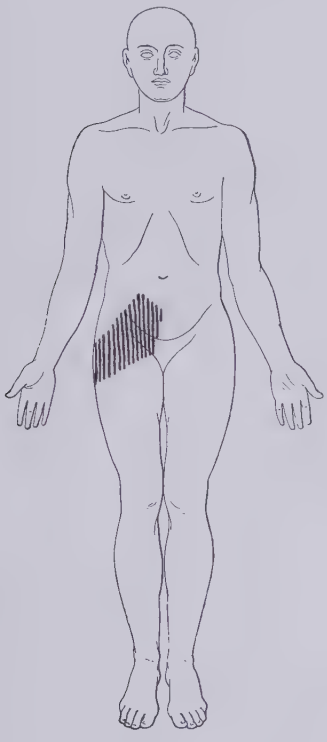


Fig. 84.

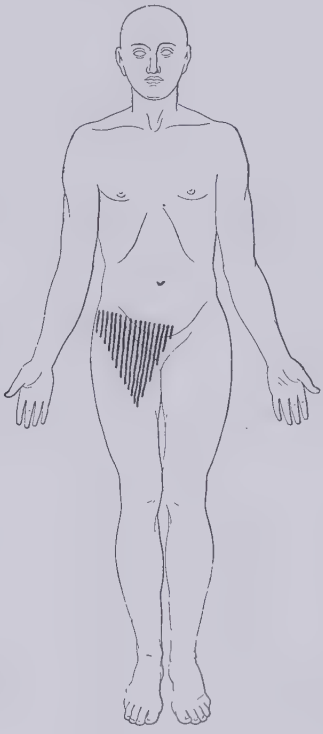


Fig. 85.

Fig. 84.—Area of cutaneous hyperæsthesia in appendicitis. (*J. Fraenkel.*)
Fig. 85.—Area of cutaneous hyperæsthesia in a case of salpingitis. (*J. Fraenkel.*)

A pain running up the back of the thigh may be caused by pressure over the back of the knee when the leg is extended at a little more than a right angle. This is diagnostic (Gowers). If the patient lies on his back and the leg is kept extended, and then the whole limb brought slowly up until it is at an acute angle with the trunk, a sharp pain in the sciatic notch is felt; this too is diagnostic. Anæsthesia over the course of the nerves occurs very rarely. When present, it indicates a severe neuritis or injury to the nerve. Muscular wasting and weakness occur after a time, and in old and severe cases partial electrical degeneration reactions

may be observed. The ankle-jerk is usually abolished, but the other reflexes remain normal or nearly so. Herpetic eruptions over the course of the nerve occur in rare cases. The affected limb usually feels colder and shows evidence of enfeebled vasomotor supply.

The disease usually lasts two or three months; not rarely it lasts six months or even a year or more. It has been known to extend slowly upward and involve the sacral plexus or even the spinal cord.

Fig. 86.—Painful joints in sciatica.

Pathology.—Only twelve autopsies are on record. There has been found in some cases a thickening of the nerve and a gelatinous exudate in the perineural sheath, the fluid not, however, containing evidence of cellular proliferation (J. R. Hunt). The arteries are thickened and sclerosed. The evidence points to the presence in ordinary sciatica of a perineuritis with exudate often of gouty or rheumatic origin.

Diagnosis.—Sciatica has to be distinguished from hip-joint disease, dislocation and sprains of the sacro-iliac synchondrosis, organic disease of the cauda equina or cord, muscular pains in the hip and leg (rheumatic myositis) and from pains caused by tumors. A true sciatic neuralgia ought also to be distinguished from sciatic neuritis. A consideration of the facts already given ought to make the diagnosis not difficult. Pure sciatic neuralgia occurs in early life and is not accompanied by much local tenderness. There is no paralysis or wasting of the limb or anæsthesia. Double sciatica is most always symptomatic of diabetes or organic disease.

Prognosis.—Almost all cases get well in from three to six months. Severe attacks in people over forty are the most intractable. Relapses occur, but not as a rule.

Treatment.—In bad cases which are seen early, the most important indication is rest. The patient should be put to bed, and the whole lower extremity secured in a splint. Ice bags or heat should then be applied

over the course of the nerve. A blue pill (gr. v.) may be given twice daily at first. In less severe or older cases the baking process may be used, or large blisters applied over the nerve in the thigh, and the application repeated in a week. If there is a rheumatic history, potassium salicylate or iodide should be given in full doses. The bowels must be freely opened. Hypodermatic injections of morphine or cocaine (gr. ¼) may be needed for a few days, the cocaine being repeated if necessary. When the disease has become more chronic, a strong galvanic current may be given daily with large electrodes, one over the lumbar region or sciatic notch, the other, which should be the positive pole, over the leg and foot. As so-called specific remedies we have oil of turpentine in doses of fifteen drops t. i. d., and this may be advantageously combined with oil of gaultheria. Massive doses of antifebrin or antipyrin sometimes stop the pains (gr. x., q. 2 h.). There are a great many local remedies which at times prove useful. Among these are mustard plasters, menthol, chloroform liniment, and the actual cautery. Bandaging the limb in sulphur to which a little menthol is added is often very efficacious. Kneading the nerve with a glass rod and an anodyne ointment is sometimes beneficial. Massage is useful after the acute stage is passed. Very little can be expected from nerve-stretching, but it may be tried as a last resort. If tried, however, great caution should be exercised in pulling on the nerve. Not over thirty to forty pounds pull should be used. The operation of cutting down on the nerve and dissecting off the sheath for a space of several inches has been tried. The injection of a normal saline solution about the nerve has been recommended.

Plantar Neuralgia.—In rare cases the pain of sciatic neuralgia is limited to the plantar nerves, and is accompanied by paræsthesia and even anæsthesia of this region. The condition here is probably a neuritis combined sometimes with arthritic changes. Erythromelalgia may be regarded as a form of plantar neuralgia.

Erythromelalgia (*red neuralgia of the feet, congestive neuralgia*) is a disease affecting both feet, and characterized by burning pains and congestion of the parts.

The disease occurs usually in men in middle life, after some infection or severe physical exertion afoot. It is due sometimes to gouty habits, and I have seen it in patients who were diabetics.

The disease begins in the ball of the foot or the heel with burning pains. The trouble increases until nearly the whole sole in the distribution of the plantar nerve is involved, and the pain, though worse at night, is almost continuous. It is much increased by exertion, the feet become very tender so that standing or walking is most painful. Meanwhile there has developed with the pain a flushing of the part upon

exertion. In bad cases the parts most affected are continuously marked by a dull, dusky, mottled redness, with some swelling. The hands may be slightly affected. Slight injuries may cause blisters and even ulcerations. The congestion usually disappears in the horizontal position and this also relieves the pain. The symptoms are worse in warm weather. The disease is very chronic and, though not dangerous to life, makes life very miserable.

Pathology.—In the cases of erythromelalgia as described by Mitchell and others, there are: (1) A vasomotor disturbance; (2) a neuritis; and (3) in rare instances, spinal-cord disease; (4) probably in most cases there is an obliterative arteritis or other form of vascular disease.

The *diagnosis* must be made from alcoholic and gouty paræthesia, podalgia, local disease of bone and ligaments, and from reflex pains.

Treatment.—Elevation of the feet and applications of cold give temporary relief. Faradization has sometimes given help; oftener it has not. There is nothing known which gives permanent relief. The physician must rely upon rest, bandaging, cold, anodyne applications, hydrotherapy and tonics. Baking the part is sometimes useful especially when there is arteriosclerosis. The salicylates, iodides, mercury, nitrites, and mineral acids with strychnine may be given.

Morton's neuralgia, so-called, is a neuralgia affecting the metatarsophalangeal joint of the third and fourth toes, and is due, it is thought, to a slight luxation, with consequent pressure on a digital branch of the external plantar nerve. It sometimes affects other toes, however. It is not always due to a luxation. Incipient flatfoot may cause it, and I have seen a typical case in a pregnant woman, disappearing after confinement. The trouble occurs generally in women, and if there is a luxation the cause is external injury or shoe pressure. The treatment is not very satisfactory. It should be directed to giving rest to the foot, and the avoidance of lateral pressure on the joints by wearing a broad-soled shoe with support to the arch of the foot. Support may also be given by a broad flannel bandage. Amputation of the toe is a very certain remedy.

Tarsalgia (*policeman's disease*) is a painful affection, due probably in most cases to an incipient flattening of the foot and stretching of the plantar ligaments. Some have ascribed it to a deep-seated contusion of the adipose cushion covering the os calcis. A chronic inflammation of the sheath of the tendo achillis causes symptoms resembling podalgia. Probably the condition varies somewhat in different cases. It is observed in persons who have been in the habit of going barefoot, and have then gone into the army or taken civil positions obliging them to stand or walk a great deal.

It was noticed originally in the policemen of Paris, and cases have

been seen in this country. The name tarsalgia was given by Duchenne.

Pododynia may be caused by a slight shortening or contraction of the tendo achillis and is relieved by stretching this tendon. Pains in the feet and legs of various types may be caused by *flat-foot* and this condition should always be carefully considered.

Treatment, medical or surgical, seems to do little for the disorder, which is very chronic. Patients are better in cold weather, and when resting the feet. Leeches, the cautery, the iodides, and broad shoes with rubber heels are serviceable.

Coccygodynia is a neuralgia affecting the lower posterior branches of the sacral nerves. It occurs oftenest in women and is caused by casual injury and labor. Coccygeal pains occur also in spinal irritation and reflexly from pelvic disease. The disease is a most annoying one, as it interferes with sitting and walking. There is often also pain at stool, and the parts are tender to pressure. The disease is usually one involving the fibrous structures of the coccyx, and is more an articular and bony than a nervous disorder. Surgical treatment, such as amputation of the coccyx, may be needed, and is sometimes effective, but not in the cases in which there is only a neurasthenia with spinal irritation, for here the trouble seems to be more a habit pain or psychosis.

PERIPHERAL VASOMOTOR AND TROPHIC NEUROSES

Symmetrical Angio-neurotic Gangrene, or Raynaud's Disease (*Abortive Form known as Digiti Mortui*).—Symmetrical gangrene or Raynaud's disease is a rare affection characterized by spasm of the vessels of the extremities, coldness, pallor, waxiness of fingers or toes, or by blueness, mottling, swelling, and pain, followed often by a dry gangrene of some of the fingers or toes.

The name is a poor one and leads to confusion. The disease is usually a peripheral obliterating endarteritis, affecting oftenest the fingers and toes, but attacking at times the nose, ears and even proximal parts of the body, such as the legs. It is an evidence of many different morbid conditions. It is seen in some types of acute rheumatism, accompanied perhaps with purpura and ecchymoses. It may be a toxic angio-neurosis.

The disease occurs usually in children and young adults. Women are affected oftener than men. Anæmia, chlorosis, and neurasthenic states predispose to it. Malarial infection, acute infectious fevers, nephritis, occupations that lead to exposure, such as washing, are causative factors. Diabetes and syphilis are also causes by leading to vascular disease.

Symptoms.—The disease comes on rather suddenly and affects oftenest two or three fingers of both hands. In its early and mild

degree there are simply a coldness, numbness and waxy pallor of the fingers. The skin looks shrunken. There is slight anæsthesia. The extremities feel as if dead. After a few hours this passes away, but returns again and may finally become an almost constant condition. Beginning in one or two fingers, it may finally involve all. The toes, tip of the nose and ears may be similarly affected, though this is rare in the milder form. Exposure to cold, even slight, is the common excitant of the lesser form of the trouble, which is commonly known as "digiti mortui," "dead fingers," or "local syncope."

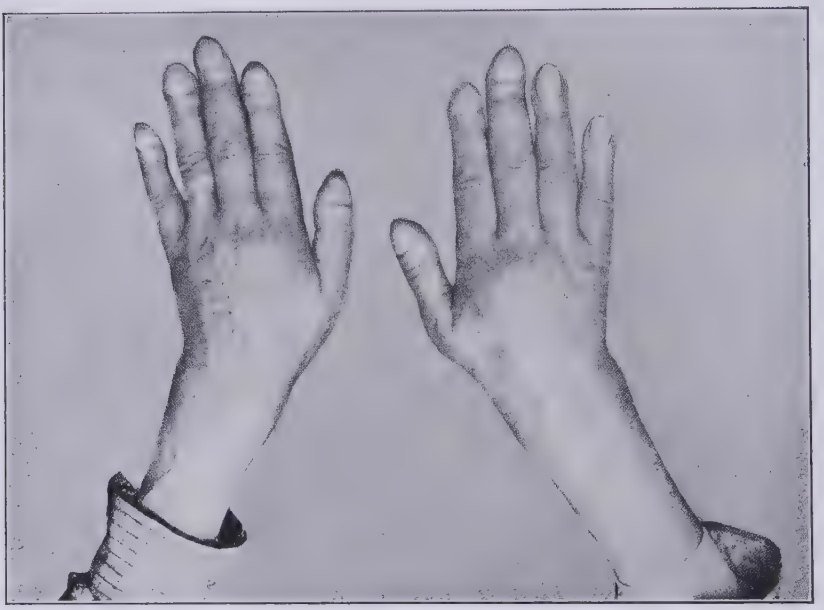


Fig. 87.—Raynaud's disease. (*Howard Fox.*)

In severer grades the fingers become blue, swollen, and there are burning sensations and much pain, but no anæsthesia. This condition is known as that of "local asphyxia," and it is usually followed by gangrene.

In the gangrenous stage small blisters appear on the distal phalanges, which fill with bloody serum, then dry up, and beneath the scab ulceration begins, which is shallow and soon heals, leaving a scar. The process then stops. In very rare cases the whole tip of the finger or toe, including the bone, becomes involved. The process as stated may attack the ears, lips, tongue and even parts of the trunk. Along with this gangrenous process there is often a hæmaturia.

The dead-finger trouble may last but a few days or weeks or it may

continue for months. The gangrenous stage lasts about three weeks. It lasts longer if it comes on in one finger after the other. The disease is one of months, and it is liable to recur.

The *diagnosis* must be made from senile gangrene, frostbite, ergot poisoning and alcoholic neuritis.

Pathology.—A neuritis has been found in some cases of so-called Raynaud's disease, but this is secondary. In most cases there has been found an obliterating endarteritis (Jacoby, Sachs). The trouble is in some cases apparently functional or at least temporary and due to the combination of an oversensitive nervous system and some irritant, such as impoverished blood, malaria, or other toxic agent, which causes spasm and probably some temporary endarteritis. In a small per cent. of cases the disease is associated with scleroderma.

Prognosis.—The cases usually get well, but the course is long and there may be relapses. In only the rarest instances has death occurred, and then from a serious and progressive arterial disease.

Treatment.—Galvanism to the spine and limbs, warm applications, anodynes, tonics, are indicated. Nitroglycerine, the iodides, chloral, may be tried. The frequent short application of rubber bandages giving a kind of massage to the vessels has been recommended. Baking the part is sometimes most efficacious.

CHAPTER XI

DISEASES OF THE SPINAL CORD

ANATOMY AND PHYSIOLOGY

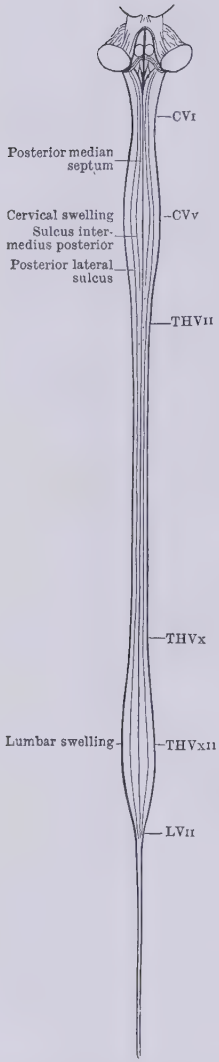


FIG. 88.—Showing the relative size of the different parts of the cord. (*Cunningham.*)

Anatomy.—The spinal cord is aslender, cylindrically shaped organ. It is from 42 to 45.7 cm. (sixteen and one-half to eighteen inches) long, being shorter absolutely and relatively in women. Its weight is about thirty-three grams (one ounce). It is suspended in the vertebral canal, where it reaches in all persons over one year of age as far down as the second lumbar vertebra. In new-born infants it extends to the third lumbar vertebra.

It is divided into cervical, dorsal or thoracic, lumbar and sacral portions, corresponding with the nerves it gives off. These are respectively four, ten and one-half, two, and one and one-half inches long (see Fig. 88). Its shape on cross-section is nearly round, except in the lower cervical region, where it is flattened antero-posteriorly. Its average diameter is 1 cm. (two-fifths of an inch). It has two swellings or enlargements, the cervical and lumbar. Their positions, size and extent are shown on the diagram. Its specfic gravity is 1.030.

It is surrounded by three membranes, all of which are continuous with the corresponding envelopes of the brain. They are the dura mater, the arachnoid and the pia mater (Fig. 89).

The spinal cord is movable up and down in its canal to the extent of from one-half to one inch.

The Nerve-roots.—The spinal nerve-roots are covered with the pia and arachnoid. They pierce the dura in two places and unite to form a mixed nerve. The dura mater is prolonged over the nerves as they pass through it, forming a tubular sheath. The anterior roots are the larger. At the point of exit of the nerves from the cord a slight constriction is formed.

The Root Ganglia.—On each posterior root, *outside the dura*, is a posterior spinal ganglion. The ganglia lie in the intervertebral canals, except those on the sacral nerves.

Fissures.—Throughout the whole length of the cord there are two median fissures, called the anterior and posterior.

Columns.—These fissures and the lines formed by the exit of the roots divide the cord into four columns—anterior, posterior and two lateral.

The Composition of the Cord.—The Cord is composed of white and gray matter. The white matter lies outside and is composed mainly of nerve-fibres, the gray matter mainly of nerve-cells. Each has also neuroglia, connective tissue,

and blood-vessels. In the gray matter is a central canal lined with epithelial cells.

The Gray matter is arranged, as shown in the figures, somewhat in the shape of a letter **H**. Its different parts are called the anterior and posterior horns and intermediate gray. At certain levels there are lateral horns. The gray matter changes in shape at different levels of the cord. It is greatest in amount at the lumbosacral junction (23.33 sq. mm.); next at the cervical enlargement (sixth cervical) (17.32 sq. mm.). It increases in amount relatively to the white matter from above downward. The gray matter of the two halves of the cord is connected by a bridge or commissure. The anterior part is composed of white medullated nerves and is called the white commissure. The posterior is composed of very fine nerve-fibres, mostly medullated

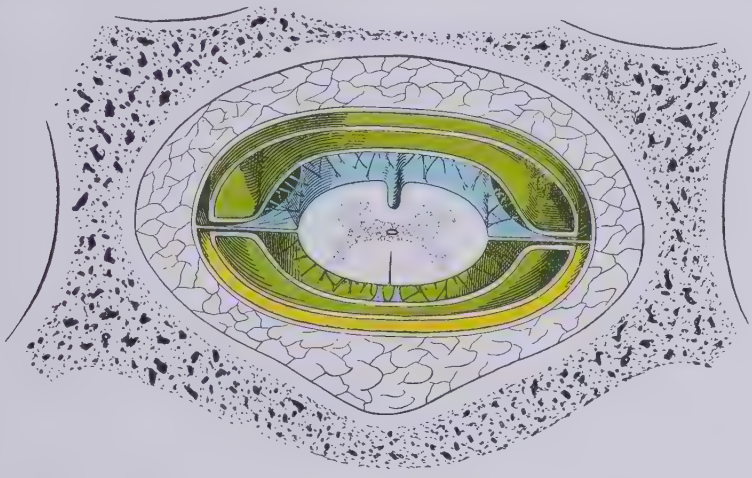


Fig. 89.—*V*, Body of vertebra; *V'*, spinous process; 1, ligament; 2, vessels; 3. dura mater with the arachnoid lying directly beneath it; 4, anterior root; 5, posterior root; 6, spinal ganglion; 7, ligament.

collaterals, called the gray commissure. Between the two is the central canal, and surrounding it is the *central gelatinous substance*, composed of neuroglia.

The posterior horns reach to the periphery. They are divided, beginning from without, into the rim zone or Lissauer's column, the spongy zone, and the gelatinous substance of Rolando. The rim zone is composed of very fine nerve-fibres; the spongy zone and gelatinous substance are composed of very small nerve-cells, some having continuous neuraxons (cells of Deiter) and some having rapidly branching neuraxons (cells of Golgi). The substance of Rolando is extremely rich in nerve-cells and is not made up of neuroglia, as was once supposed.

To sum up, we have the gray matter—

Arranged in:		Composed of:
1. Horns	anterior, lateral, posterior.	*a*. A ground substance of neuroglia and connective tissue forming the substantia spongiosa.

2. Intermediate gray.
   - *b.* Cell groups: internal, anterior, etc.
   - *c.* Plexuses of fine nerve fibres, *e.g.*, in the rim zone.
   - *d.* Masses of neuroglia:
     - (1) The central gelatinous substance.
     - (2) The periphery of the cord and the spongy zone.
   - *e.* Blood-vessels and connective tissue.

Now, taking up some of these factors in detail, we find that:

(*a*) The ground substance of the gray matter is made up of a fine mesh-work of fibres which are the processes of neuroglia cells and of nerve-cells. Besides this, there is some connective tissue, and there are prolongations from the base of the epithelial cells lining the central canal.

(*b*) The nerve-cells are, in part, arranged in groups with the long axis parallel to that of the cord. The cells are surrounded by a rich plexus of dendrites and end brushes, as well as by the supporting neuroglia matrix, a little connective tissue, and many small blood-vessels. The cell groups are named in accordance with their position—internal, antero-lateral, lateral median, posterior or sensory cells, and the cells of Clark's column (Fig. 90).

This nomenclature answers for ordinary anatomical descriptions. Histologically, we find two kinds of cells, the root cells and column or tract cells (Strangzellen). The former are cells whose neuraxons pass out to form the anterior roots. They form the great part of the anterior horns. Deep in the anterior horns are a few root cells, whose neuraxons pass into the posterior roots and hence to the ganglionic system. The column cells are found in the posterior horns, intermediate gray and to some extent in the anterior horns. Their neuraxons pass to the white matter of the same or opposite side, and furnish commissural, associative and even long column fibres.

The anterior-horn root cells are arranged in groups, which overlap each other. Each group has the special duty of presiding over certain sets of muscles or other organs which have a common function. These cells are large in size, 35 to 100 μ $\frac{1}{700}$ to $\frac{1}{250}$ in.); they are multipolar, having five or six processes, one of which is an axis-cylinder process, which, in lower animals at least, gives off a collateral before it leaves the cord. The cells in the central parts of the horn are the smaller; the cells in the lumbar swelling are largest, because they are connected with long nerves. The cells of the cervical swelling are next in size. The cells of the posterior horn are small and multipolar. The cells of Clark's column are bipolar, 30 to 60 μ ($\frac{1}{800}$ to $\frac{1}{400}$ in.) in diameter, and are arranged with their long diameter parallel to the axis of the cord. They are grouped together in a kind of nest at the inner and central part of the posterior horn (see Fig. 90). Clark's column is most distinct from the eighth dorsal to the second lumbar nerves, but extends up as far as the last cervical. An analogous group of cells is found at the level of the second and third sacral nerves. A small group of spindle-haped cells lies in the intermediate gray matter at the base of the posterior horns. There are other minor groups of cells which it is not necessary to describe here.

The white matter of the cord is composed mainly of neuraxons and the collaterals of these running in a supporting network of neuroglia, connective tissue and blood-vessels. Surrounding it and lying just beneath the pia mater, is a thin layer of neuroglia 5 to 50μ ($\frac{1}{5000}$ to $\frac{1}{30}$ in.) thick. The neuraxons are medullated but have no neurilemma, and but few, if any, nodes of Ranvier. There are two kinds: the large (81 to 20μ) and the small (2 to 3μ) in diameter. The small fibres make up the postero-internal (Goll's) column entirely, and are numerous in the deep part of the lateral columns, but they are found in all regions. The fibres run up and down for the most part, but constantly send off branches to the gray matter. They are arranged in col-

umns, the division being based partly on anatomical, partly on physiological, and partly on embryological grounds.

Anatomically, there is a simple and natural division, which we have already given, into the anterior, lateral and posterior columns, the divisions being made by the median fissures and the roots of the nerves.

On physiological and embryological grounds the columns are further subdivided as follows:

- The anterior columns are divided into
  - Direct pyramidal tract.
  - Anterior fundamental column.
- The lateral columns are divided into
  - Lateral fundamental column.
  - Lateral limiting layer.
  - Crossed pyramidal tract.
  - Direct cerebellar tract.
  - A ventral cerebellar tract, or Gowers' tract.
- In the anterior and lateral columns the principal tracts are:
  - Rubro-spinal tract.
  - Vestibulo-spinal tract.
  - Tecto-spinal tract.
- The posterior columns are divided into
  - Postero-internal column, or column of Goll.
  - Postero-external columns. or column of Burdach.
    - Burdach's column is divided into
    - Middle root zone.
    - Posterior root zone.
  - The ventral zone.
  - The comma.
  - The oval zone.
  - The triangular column.
  - Rim zone, or column of Lissauer.

The fibres which make up these columns are of two kinds—*long* or projection, *short* or associative.

The long fibres connect the different levels of the cord with the brain, and the posterior spinal ganglia with nuclei in the upper part of the cord.

The *short* or associative fibres connect different levels of the cord with each other, and also connect the two halves of the cord at the same levels. The long-fibre tracts lie on the periphery of the cord, as a rule; the short-fibre tracts lie near the central gray.

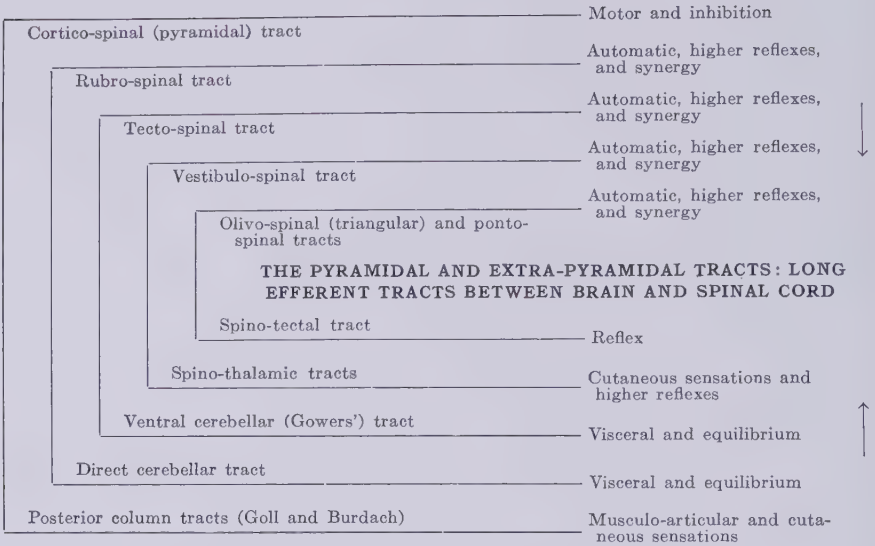
The names of the long-fibre tracts are the direct and crossed pyramidal, the direct cerebellar, the antero-lateral ascending, or ventral cerebellar, the rubro-, vestibulo-, and tecto-spinal tracts, the postero-internal or column of Goll and the upper part of the column of Burdach.

The *direct pyramidal tract* lies along the anterior median fissure and extends down as far as the lower part of the dorsal cord. Its fibres cross over in the anterior commissure at various levels and connect with the cells of the anterior horns.

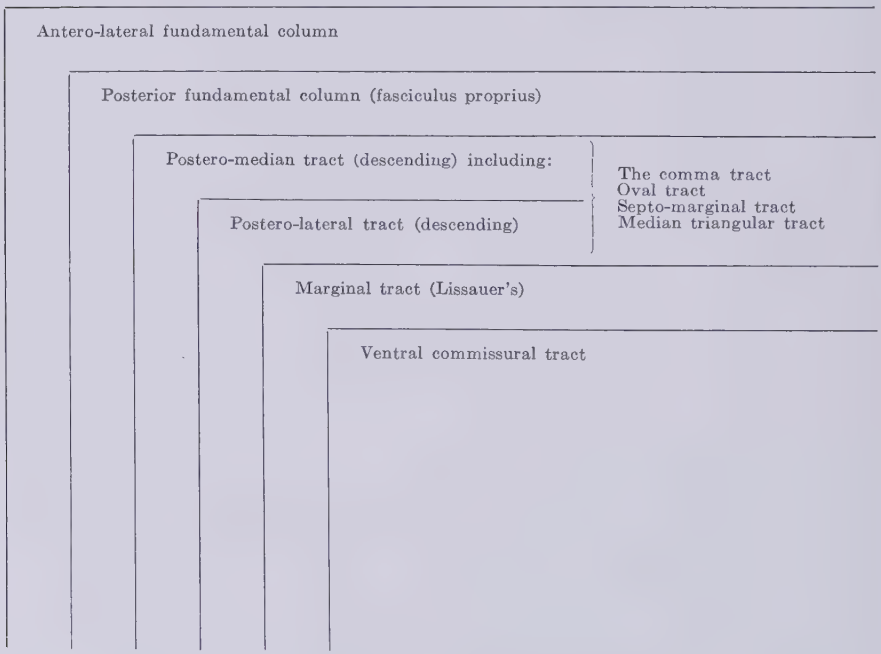
The *crossed pyramidal tract* extends down the whole length of the lateral column of the cord and sends its fibres to the anterior horns of the same side.

Both of the above tracts are continuations of the anterior pyramids or motor tracts of the medulla. These pyramids divide at the lower end of the medulla, about 90 per cent. of fibres crossing over to form the crossed pyramidal tract and 10 per cent. continuing on the same side. Some of the fibres of the crossed pyramid redecussate (in lower animals) and enter the pyramidal tract of the side on which they started.

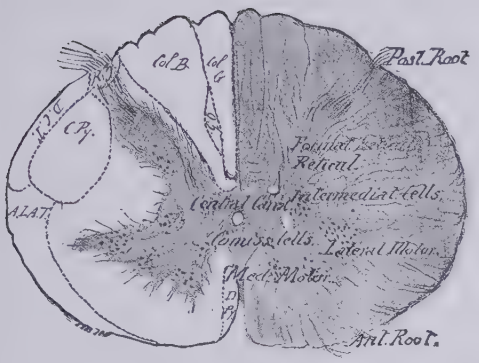
The **extra-pyramidal tracts,** are tracts of efferent fibres which have to do with motion, tonus, and the higher automatic actions.



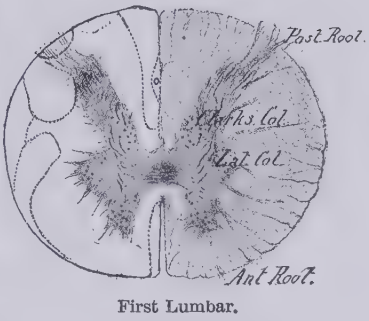
THE LONG AFFERENT TRACTS BETWEEN THE BRAIN AND THE SPINAL CORD.



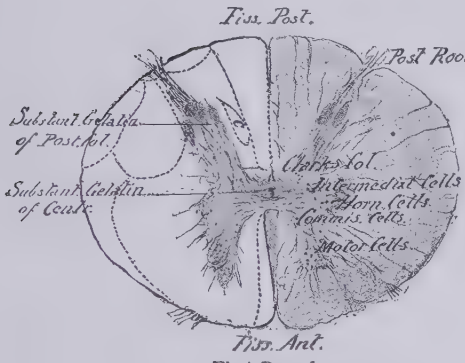
THE SHORT OR ASSOCIATION TRACTS OF THE SPINAL CORD.



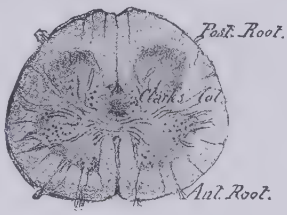
Sixth Cervical Segment.



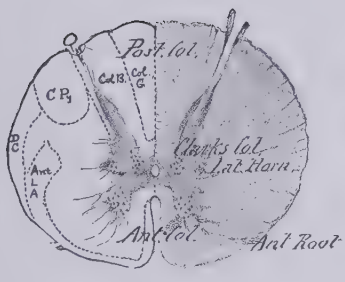
First Lumbar.



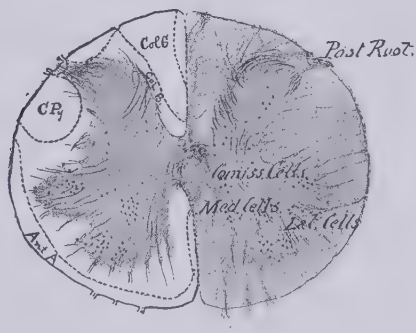
First Dorsal.



Fourth Sacral.



Eighth Dorsal.



First Sacral.

FIG. 90.—Showing the arrangement of the gray and the white matter at different levels of the cord, also the columns and cell groups.

The *direct cerebellar tract* begins at the level of the first lumbar nerve. Its fibres originate in the vesicular column of Clark. They pass up to the cerebellum and go chiefly to the cortex of the vermis without crossing.

The *ventral cerebellar* tract, or column of Gowers, extends nearly the whole length of the cord. Its fibres come from the anterior commissure and the sensory cells of the opposite posterior cornu. They pass through the medulla and pons, turn and enter the cerebellum to end in the cortex of the vermis. A few fibres pass directly to the optic thalamus.

The *rubro-spinal tract* originates in the red nucleus; it crosses to the opposite side, passes down through the pons and medulla, runs in the lateral column of the cord forming a triangular or wedge-shaped tract along the anterior or ventral part of the

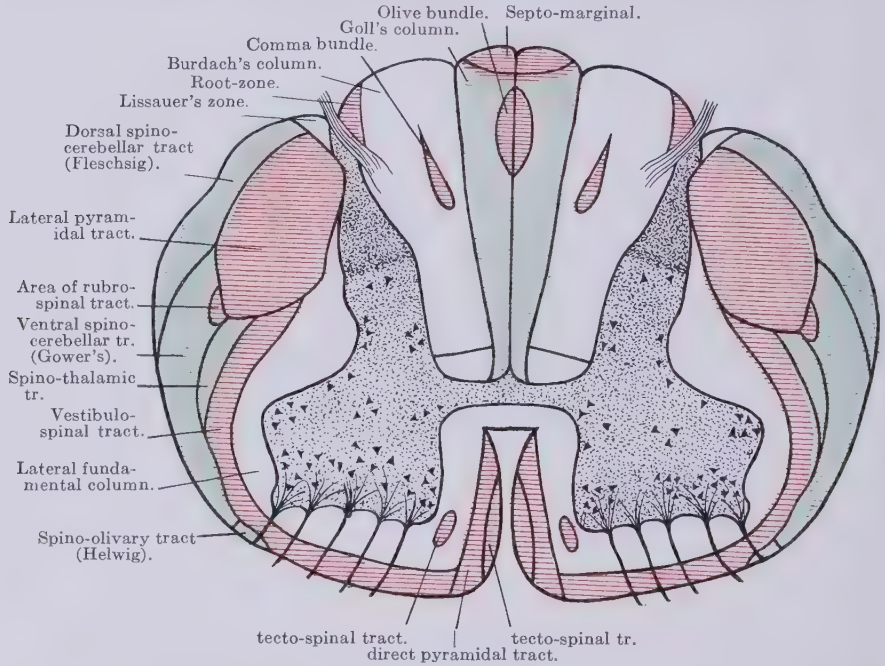


FIG. 91.—Showing the tracts and columns of the spinal cord. Descending tracts are in red; ascending in green. The vestibulo-spinal is not as extensive as indicated and lies mainly in the ventral column. The rubro-spinal tract is not as sharply defined as indicated.

crossed pyramid tract. It extends as far down as the sacral cord. Its fibres end in the anterior horns.

The *vestibulo-spinal tract* arises in Deiter's nucleus, crosses and runs in the lateral column of the cord in front of the rubro-spinal. It may extend to the anterior column. It passes down as far as the lumbar cord. Its fibres end in the anterior horns.

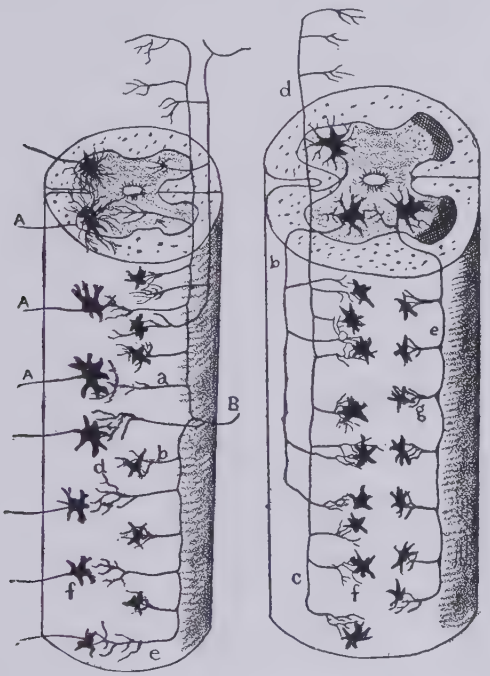
The *tecto-spinal tract* originates in the corpora-quadrigemina and takes a similar course to the preceding tract, a median part running in the anterior and a lateral in the lateral columns.

Several other tracts are described. These together with the above three small tracts degenerate downward and are spoken of collectively as the extra-pyramidal tracts.

The posterior columns are made up of fibres that come from the posterior spinal ganglion and pass directly into the posterior columns, then ascend to nuclei near the medulla where they end. These fibres are called exogenous. The columns also receive some fibres from the tract cells in the posterior horns; and they are called endogenous fibres.

The exogenous fibres in the sacral lumbar and lower dorsal cord pass inward and form the postero-internal column or Goll's column. This is then small below and larger in the cervical region. They end in the nucleus of Goll's column (nucleus gracilis).

The exogenous fibres in the upper part of the cord pass up, lying to the outer side of the column of Goll. They form the column of Burdach and end in the nucleus of Burdach (nucleus cuneiformis).



I Fig. 92. II

I. Showing the connections of the anterior and posterior roots and cornua with each other (Cajal). *A*, anterior root; *B*, posterior root; *a*, collaterals; *d*, end-brushes.

II. Showing the association or short-fibre system of the cord (Cajal). $a,^{r}$ anterior cornua cell; *b*, *c*, *d*, association fibres; *e*, posterior association fibres.

The column of Burdach is thus not distinctly formed as a long fibre tract except in the upper part of the cord.

The names of the short-fibre columns are the anterior and the lateral fundamental columns, the lateral limiting layer and the lower part of the column of Burdach.

The posterior columns also contain three short-fibre columns whose cells of origin lie in the gray matter of the cord. The fibres in the cervical region lie in the shape of a comma (comma of Schultze), in the lumbar region in the shape of an oval (oval zone of Flechsig), in the sacral zone in the shape of a triangle (triangle of Gombault). Besides this there is a zone of short fibres lying close to the gray commissure the whole length of the cord—ventral zone or posterior fundamental column.

The Relations of the Different Parts of the Spinal Cord to the Peripheral Nerves, and with Each Other.—I will begin with a description of the way in which the anterior and posterior nerve-roots are conected to the cord; then describe the mode in which the different columns and cell groups are connected with each other; and finally I will indicate briefly the connections of the cord with the brain.

The *anterior nerve-roots* are connected directly with the anterior-horn cells, of which they are processes, and together with which they form the peripheral motor neurons. It is possible that in man they send off collaterals before leaving the cord.

The cells of the anterior horn are surrounded by two chief sets of "end brushes," one coming from the pyramidal and other descending tracts, the other from the posterior horns and roots. Thus these cells are in relation with impulses from the brain and from the periphery.

The *posterior nerve-roots* originate in the cells of the spinal ganglia. These give an axon which divides and sends off a peripheral branch to form the sensory nerve, and a central branch which passes into the spinal cord forming the posterior root. On entering the cord the fibres of the posterior root divide like a T and pass up for one or more inches and down for a short distance only. These root-fibres, with their cells of origin and the sensory nerve-fibres, form together the *peripheral sensory neurons.*

The peripheral sensory neurons send their axons into the spinal cord through the posterior roots, and these axons divide, run up and down the cord for a short or long distance and then end in terminal arborizations about different groups of cells.

1. Some pass directly into the columns of Goll or Burdach on the same side and pass up to end in the nuclei of these columns.
2. Some pass to cells in the posterior horns of both sides and there end.
3. Some pass to cells of the column of Clark of the same side.
4. Some pass to cells of the anterior horns of both sides.[1]

[1] There are, according to Ingbert, about 650,000 sensory fibres in the posterior spinal roots; each cutaneous fibre supplies from 1 to 3 sq. mm. of surface.

The different parts of the spinal cord are connected by the short fibres which unite different levels of the cord, and by commissural fibres which unite the different halves of the cord. These short and commissural fibres originate in groups of nerve-cells lying in the central parts of the gray matter and which we have called column nerve-cells. They are small and multipolar. Some are distributed sparsely in the white columns. Fibres arise from them, run in the commissures and short-fibre tracts, and end in brushes which put the fibres in relation with various cell groups.

DIAGNOSTIC PHYSIOLOGY

General.—The spinal cord is made up of (1) peripheral motor and sensory neurons; (2) the upper or encephalo-spinal neurons; (3) short associative neurons. Lesions of the peripheral motor neuron cause motor irritation or a severe, flaccid paralysis and atrophy. Lesions of the peripheral sensory neurons cause pain, paræsthesia, ataxia and anæsthesia and secondary trophic troubles. Lesions of the upper or cerebrospinal neurons cause a spastic paralysis without atrophy. Lesions of the secondary sensory neurons cause little pain or sensory irritation, but lead to anæsthesia paræsthesia, ataxia, etc.

The relations of the spinal cord to the brain will be described in connection with the anatomy of the brain.

Peripheral Palsies; Radicular or Root Palsies; Nuclear Palsies.—If the nuclear groups of motor cells in a single segment of the spinal cord are destroyed, *e.g.* the fifth cervical segment, we get a partial paralysis of the muscles supplied from this segment, because these nuclear groups overlap and are not each assigned to a single segment.

If an anterior root is cut across, we also get a partial paralysis, because muscles generally are supplied from several different roots as well as from several different segments. Hence a single segment or *root palsy* is rarely a complete one. On the other hand, a section of a motor nerve may cause a complete paralysis of a muscle or group of muscles. Hence a *peripheral* palsy may be complete.

If the entire nuclear group of cells controlling a muscle-group is destroyed, as happens in anterior poliomyelitis, we also get a complete paralysis—*nuclear palsy*.

Sensory Ansæthesia from Peripheral, and Radicular Lesion.—In the same way we get peripheral anæsthesia over a definite cutaneous area when a peripheral sensory nerve is cut.

When one posterior root is cut we get no anæsthesia; when, however, two or more are cut we get bands of anæsthesia known as radicular or segmental zones. These have been determined by animal experiment and operations and disease, so that we have charts showing the radicular zones produced by cutting two posterior roots at different levels. They are marked singly for purposes of convenience, only (see Figs. 27 and 28).

Automatic Centres.—The nerves and cells of the cord are arranged in complex groups which preside over certain functions or respond in a definite way to certain stimuli. These are called the spinal automatic centres. They are the cilio-spinal, secretory, vasomotor, genital, vesical and rectal. The important parts of these centres lie deep in the gray matter on either side of the central canal, but nearer the base of the posterior horns. Lesions of the white matter or of the anterior or posterior horns do not directly affect them.

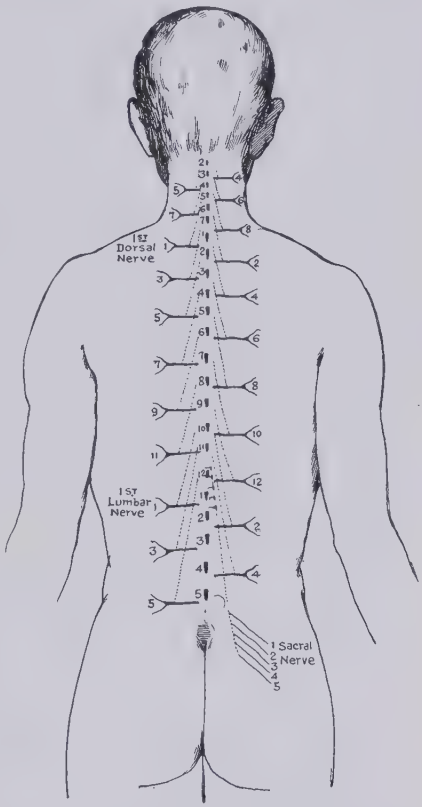


Fig. 93.—Showing the relation of the spinous processes to the points of origin in the cord of the spinal nerves. The nerves are shown in black lines as they come from the spinal canal. The dotted lines show the level at which each nerve arises in the cord.

The *cilio-spinal centre* lies in the eighth cervical and the first dorsal segment. Its stimulation causes the pupils to dilate.

The *genital centres*, including those for erection and ejaculation, reach from the first to the third sacral segment, inclusive.

The *bladder and rectal centres* are in the fourth and fifth sacral segments, extending up and down a short distance, the bladder being perhaps a little lower.

The spinal *vasomotor centres* extend from the second dorsal to the second lumbar segments. The vasodilator nerves pass out by the anterior, the constrictor by the posterior root (Gaskill).

The *cells of the posterior horns* are sensory in function and are connected with the tactile, pain, temperature and reflex fibres of the posterior roots.

Topography and Localization.—The neurologist and surgeon need to know, for purposes of diagnosis:

1. The relation of the spinal nerve-roots, at their point of origin, to the spinous processes. This is shown in Fig. 93. In general it will be seen that the different pairs of nerve-roots arise opposite the spinous process of a vertebra one or two segments above those between which it makes its exit. Thus the sixth cervical originates opposite the fourth cervical spine, the sixth dorsal between the third and fourth dorsal spines, the first lumbar between the eleventh and twelfth dorsal spines. There is considerable variation in these relations.

2. The next point desired is the special function of each pair of nerve-roots anterior and posterior, and the level of the various centres in the cord. This is shown in the following table, based on that originally devised by Starr, modified by Mills, Sachs and myself from personal experiments and the clinical and pathological observations of Thorburn and others. (See Appendix.)

The blood-supply of the spinal cord is a subject of great practical importance; and I shall present the matter here in some detail.

The spinal cord is supplied with blood by branches from the vertebral, ascending cervical and superior intercostal arteries above, and by the dorsal intercostal, lumbar and sacral arteries below. These send off small branches which enter the spinal canal through the foramen magnum above and the intervertebral foramina at the sides: they pierce the dura mater and are distributed on the pia mater and in the cord. The arteries that thus supply the cord are these:

Primary Arteries	Origin from	Ending in
Anterior spinal...	Vertebral (from subclav.).....	Anterior median spinal artery.
Posterior spinal..	Vertebral.	
Lateral spinal....	Vertebral.	
Lateral spinal....	Ascending cervical (from subclav.).	Anterior and posterior spinal root arteries.
Lateral spinal....	Superior intercostal (from subclav.).	Anterior and posterior spinal root arteries.
Lateral spinal....	Thoracic intercostal (from aorta).	Anterior and posterior spinal root arteries.
Lateral spinal....	Lumbar (aorta). Lateral sacral (from int. iliac).	Anterior and posterior spinal root arteries.

The anterior spinal arteries are branches of the vertebrals. They unite to form the anterior median artery, which runs down the whole length of the cord, receiving re-enforcements from the lateral arteries (Fig. 94). The *anterior spinal* arteries themselves nourish only a few upper segments of the cord. The anterior median artery is not, as has been taught, a true prolongation of the anterior spinals, but is really made up by the lateral spinals. In other words, the vertebral artery through its branches nourishes only the upper cervical region of the cord. The *posterior spinal* arteries are smaller than the anterior and unite on the posterior surface of the cord. They do not continue down as a posterior median artery—there is no such artery; but they help to form two plexuses on the postero-lateral surfaces of the cord.

The *lateral spinal* arteries are derived from branches of the subclavian artery as far down as the second dorsal root; below this point by the thoracic and abdominal aorta and the internal iliac. It is an interesting fact that at or a little below the point

where the blood-supply changes from the subclavian above the heart to the aorta below, pathological disturbances frequently occur (transverse myelitis).

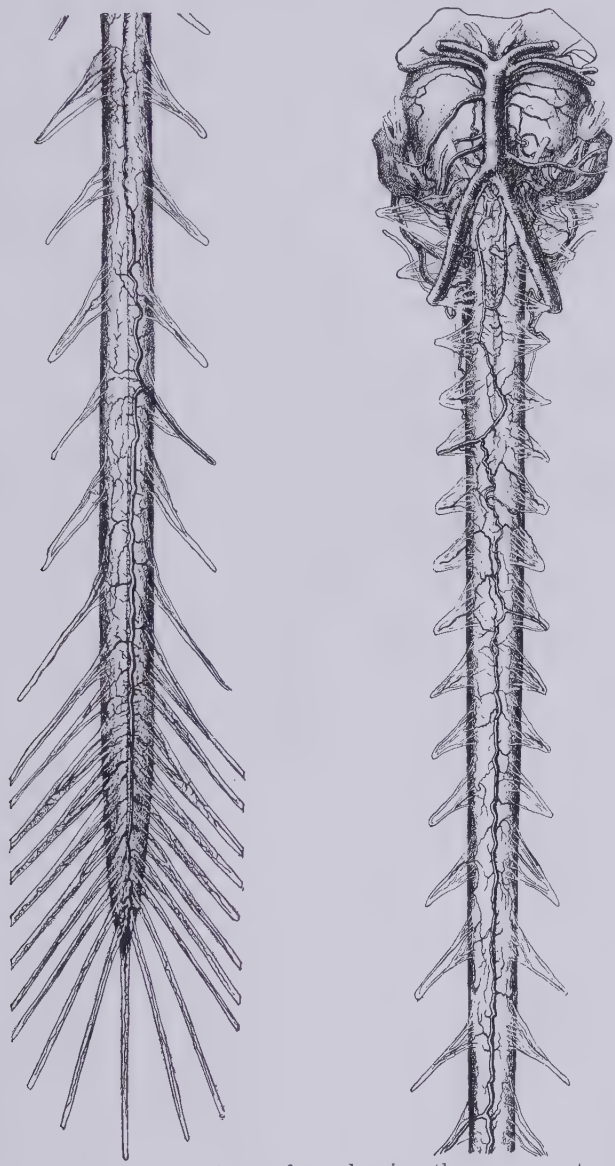


FIG. 94.—The spinal cord, anterior surface, showing the nerve roots, root arteries, and anterior plexus. (*Kadyi.*)

Root Arteries.—The lateral spinal arteries, after they enter the spinal canal, are called the root arteries. They pierce the dura mater and pass, some along the posterior and some along the anterior roots, to the cord. There are about eight

anterior-root arteries (five to ten) and about sixteen posterior-root arteries (see Figs. 94, 95). The anterior arteries are twice as large (1 mm. in diameter) and one-half as numerous as the posterior. The root arteries of the cervical region are rather the more numerous. There is a large and constant anterior-root artery in the dorso-lumbar region. The last two lumbar, the five sacral nerves and the unpaired coccygeal nerve when it exists, are accompanied by small root arteries which do not reach up to the cord itself. The lower part of the spinal cord is supplied by large root arteries from the lateral spinal arteries. Hence, the theory of Moxon that the circulation here is feeble is not supported by Kadyi's investigations.

The Plexuses.—The anterior root arteries pass to the anterior median fissure, and then divide, partly to form the anterior median artery and partly to form a rich plexus between the anterior roots; this is called the *anterior arterial plexus.* The posterior root arteries subdivide before they reach the cord, and send twigs to its lateral and

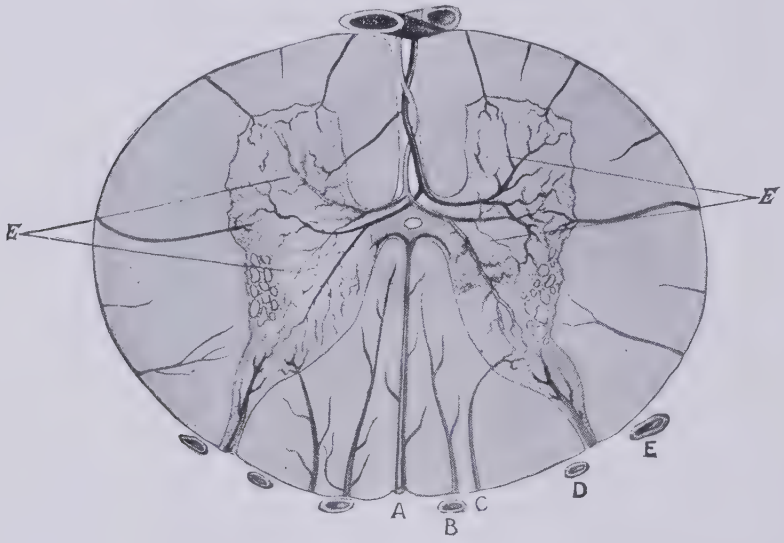


Fig. 95.—The arterial supply. *A*, The artery of the posterior fissure; *B*, the interfunicular; *C*, artery of posterior horn; *D*, of posterior root; *E*, of postero-lateral column.

posterior surfaces which form the *postero-lateral arterial plexus.* The posterior-root arteries do not anastomose to any extent with each other or form a posterior spinal artery, as is done by the anterior-root arteries. There are, therefore, three relatively independent arterial plexuses: the anterior plexus, the two postero-lateral plexuses.

Veins.—The veins of the spinal canal outside the dura mater have valves, those within it have none. The veins reach the pia mater and cord by passing along the nerve-roots. Hence, we have anterior and posterior *root veins*, corresponding to the root arteries, but more numerous, there being a total of forty or fifty. The anterior-root veins are more numerous than the posterior, but smaller (twenty five to twenty). The veins are a little larger than the arteries, the anterior veins being ½ to 1 mm., the posterior 1½ to 2 mm., in diameter.

Thus we see that the posterior surface of the cord has more and smaller arteries, fewer but larger veins. The posterior surface is on the whole more richly supplied

with veins, the anterior surface with arteries. The lateral surfaces are the least vascular.

Anterior root arteries	5 to 10	1 mm.
Anterior root veins	25 to 30	½ to 1 mm.
Posterior root arteries	16	½ mm.
Posterior root veins	20 to 25	1½ to 2 mm.

Vessels of the Cord Substance.—The cord is supplied by (1) central arteries which are branches of the anterior median, and by (2) peripheral arteries which come from the plexuses on the pia mater. These two systems have been called also the centrifugal and centripetal, respectively. They are not absolutely independent, but are in a good measure so. The central arteries nourish chiefly the gray matter, the peripheral arteries the white. Both systems are made up of "end arteries;" *i.e.*, they do not anastomose with each other. Neither the central nor the peripheral arteries are distributed in accordance with anatomical relations or physiological functions. Each cell group, for example, has a vascular supply from several sources.

The *central arteries* are given off from the branches of the anterior median at the bottom of the median fissure and number about two hundred, each spinal segment having six or seven. The accompanying central veins are small and their total capacity is less than that of the arteries, so that the central arterial pressure must be high, on account of the poor venous outlet (Kadyi). Some of the blood escapes by the peripheral veins.

The *peripheral* arteries pass into the spinal cord for the most part along the various connective-tissue septa. There they branch and supply chiefly the white matter. They supply the apex and some of the deeper substance of the posterior horns and Clark's columns. The arteries of the posterior septum are the largest and most numerous, often reaching to the gray commissure. The peripheral arteries are smaller than the corresponding veins (0.04 to 0.2 mm.). The relation is just the reverse, therefore, to that of the central arteries and veins. The peripheral arteries are small, and after passing into the cord branch into minute vessels which pass up and down and soon become capillaries. The central arteries, on the other hand, continue large, and run up and down some distance before they are subdivided into capillaries.

To sum up: The arteries predominate in total capacity in the anterior plexus and central arteries; the veins in the posterior plexuses and peripheral vessels. The central arteries are larger and longer than the peripheral. Hence the blood circulates more quickly and under greater pressure in the central gray of the cord. Conditions of enfeebled circulation would affect the posterior columns and roots more than the anterior and central parts of the cord.

CHAPTER XII

THE DISEASES OF THE SPINAL CORD

There are about thirty diseases which may be classified as belonging to the spinal cord. Most of these are organic in character and come under the head of inflammatory and degenerative or system diseases. Functional disorders referable to the cord alone are rare; while of organic diseases, those that result from injury, syphilis and inflammation are the most common.

The special diseases of the spinal cord are the following:

1. *Malformations.*—Myelocele, meningo-mylocele (spina bifida), meningocele, heteropia, amyelia, micromyelia, macromyelia, double cord.

2. *Vascular Disorders.*—Anæmia, hyperæmia, hemorrhage, endarteritis with aneurism, embolism or thrombosis, œdema. *Secondary* to these conditions are *softenings* and *sclerosis.*

3. *Inflammations.*—Meningitis, myelitis, multiple sclerosis. Secondarily, softenings, sclerosis.

4. *Degenerations or Parenchymatous Inflammations.*—Primary: posterior and combined scleroses, hereditary scleroses, progressive muscular atrophy and allied types.

5. *Tuberculosis.*—Miliary, solitary, and meningeal, causing 2, 3, 7.

6. *Syphilis.*—Which may cause 1, 2, 3, 4 and 7.

7. *Tumors.*

8. *Functional and toxic disorders.*

MALFORMATIONS

Spina Bifida (Rhachischisis Posterior)

Spina bifida is a congenital hernia of the spinal membranes, and sometimes of the cord, through a cleft in the vertebra caused by absence of the vertebral arches. It is often really a malformation of the vertebral canal rather than of the cord, though both conditions may be present.

Etiology.—The condition is not very rare, about one child in 1,200 (French statistics) being affected. It is often associated with hydrocephalus or with some other defect in development, such as ventral hernia, imperforate anus or pharynx. Hereditary influence is sometimes a factor. It is a true developmental defect, and is not due to a primary dropsy of the cord, as was once taught. It occurs rather oftener in females.

Forms.—There are three varieties described:

1. Spinal meningocele is a condition in which the spinal membranes alone protrude into the sac.

2. Spinal meningo-myelocele is a form in which the membranes and cord both protrude.

3. Syringo-myelocele (hydrorrhachis interna) is a form in which the fluid is in the central spinal canal, and the inner lining of the sac is formed by the meninges and thinned-out spinal cord.

Anatomy.—The first two forms are the most common and are called hydrorrhachis externa. The fluid here lies in the subarachnoid sac, and hence the wall of the protruding cyst is lined with the dura and arachnoid. The nerves and cord protrude into the sac in two-thirds of the cases (forming a meningo-myelocele), but in some of these only a few nerves are found. These structures, when present in the sac, as in men-

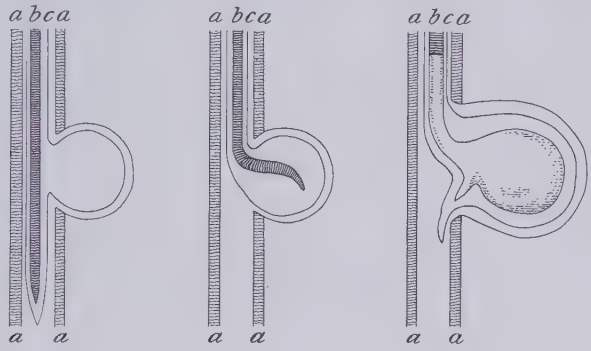


FIG. 96.—Meningocele. Meningo-myelocele. Syringo-myelocele. *a*, Vertebral walls; *b*, cord; *c*, membranes.

ingo-myelocele, lie on its *posterior and median surface.* They are attached to and form part of the wall. The spinal nerves therefore start from the wall of the sac and go back into the vertebral canal. The tumor contains cerebrospinal fluid, and occasionally connective tissue and fat (Fig. 97). The external surface is often red and smooth, and there is sometimes a depression on its median surface where the cord is attached.

Symptoms.—Spinal bifida occurs almost always in the lumbar and sacral region, the reason being that the laminæ here are the last to solidify. Usually but two or three vertebræ are involved.

The tumor varies in size from 3 cm. (1 in.) to 15 cm. (6 in.) in diameter, and may have a broad base or be pedunculated. The outer skin is often glossy, or tough, thickened or ulcerated.

Children with spina bifida are usually feeble, badly nourished and poorly developed mentally. Paraplegia occurs in half the cases, sometimes with anæsthesia and involvement of the sphincters. Talipes occurs quite often.

The *prognosis* is grave. Most subjects die unless treatment is applied, and even then the prospect is not very good. The prognosis is best for meningocele.

The *diagnosis* is easy. It is generally only necessary to exclude congenital tumors which happen to be located in the lumbo-sacral region. The most important question to decide is whether the cord and nerves are present in the sac. This may be assumed as probable if there is much paraplegia, anæsthesia and sphincter trouble, and if there is a depression on the median external surface. The introduction of an insulated needle connected with an electric battery may be tried.

The *treatment* is strictly surgical, and then is of avail only in meningocele. Injections of Morton's fluid (iodine, gr. x.; potas. iodid., gr.

Fig. 97.—Spina bifida.

xxx.; glycerin, ℥i. Dose, ℥i) have been successful. These injections should be made in the lateral portion of the sac, and the child should be kept on the back. Puncture and withdrawal of fluid with compression is not a justifiable operation. Ligaturing or opening and excising of the sac are dangerous, especially if, as is often the case, part of the cord and nerves lie in the sac. In recent years, surgical results have been more favorable and warrant serious consideration. No surgical treatment should be attempted, however, until two or three months after birth.

OTHER MALFORMATIONS

Heteropia is a rare malformation in which masses of gray matter are found in abnormal situations. A false heteropia may be caused by manipulation of the cord in its removal after death. The displaced masses consist of nerve-cells or neuroglia.

Amyelia, or absence of the spinal cord, can exist only when the brain is absent; but absence of the brain may occur without absence of the

cord. In amyelia the spinal nerves are usually present. Amyelic monsters cannot live.

Double cord is a very rare defect and involves only part of the cord except in cases in which there is a double vertebral canal. It occurs sometimes in connection with spina bifida.

Double central canal is not rare. It usually involves only a part of the cord. The two canals are side by side.

Asymmetry of the cord, usually due to abnormality in the course of the pyramidal tracts, is not extremely rare.

Splitting of the cord and defects in development at special levels are occasionally observed.

Micromyelia is a condition in which the spinal cord is abnormally short or small in size, and is not a very rare anomaly. The normal adult cord has a diameter in its various parts of 6 to 9 mm. (dorsal), 8 to 11 mm. (upper cervical), 15 mm. (cervical swelling), and 12 mm. (lumbar).

SPINAL HEMORRHAGE (SPINAL APOPLEXY)

This general name may be given to (1) spinal meningeal hemorrhage or hæmatorrhachis, and (2) hemorrhage into the cord substance, or hæmatomyelia.

1. Spinal meningeal hemorrhage is far the most common form. It may be outside or inside of the dura, the former being rather oftener seen.

Etiology.—It occurs in newly born children and in adults, and is more common in men than in women. Injuries, falls, fractures of the spine are the most frequent exciting causes. Severe convulsions from epilepsy, eclampsia, tetanus, chorea or strychnine and even severe muscular exertion may be a cause. Childbirth, purpura and the blood states following malignant infectious fevers, bursting of an aortic or vertebral aneurism, and cerebrospinal meningitis are rare causes.

Symptoms.—In small hemorrhages there may be no symptoms. In large effusions there are sudden very severe pains in the back, extending into the limbs with numbness, tingling, hyperæsthesia and muscular spasm, especially of the back muscles. Later there may be weakness or paralysis and anæsthesia of the extremities, with disorder of the visceral centres. The reflexes are exaggerated. The distribution of the anæsthesia may be segmental unless the hemorrhage is very large. The symptoms reach their height usually in a few hours. Then amelioration may occur, followed by slow recovery or with symptoms of chronic meningitis. Rarely death occurs early from exhaustion.

Diagnosis.—A history of injury or childbirth, sudden onset of attack, with symptoms of pain and meningeal irritation with rigidity which rather rapidly subside, point to extra-dural hemorrhage. In hæmatomyelia there are less pain and irritation, but more profound paralysis and

anæsthesia. The same is true of crush of the cord from fracture or dislocation. In tetanus there is a slower development of the symptoms and trismus is present. Lumbar puncture will assist the diagnosis.

The *prognosis* is grave in severe cases, but if the patient survives three or four days the prospect of partial or nearly complete recovery is good.

The *treatment* is perfect rest in bed and the administration of remedies to move the bowels and relieve pain; leeches and other local applications are of doubtful value. If there is distinct evidence of fracture or dislocation surgical interference may be indicated. It is of no use to give styptics except in purpura, when mineral acids or suprarenal extract may be tried. Later, one may give iodide and mercury and use blisters to the back.

2. **Hemorrhage into the Substance of the Cord (Hæmatomyelia)**—*Etiology.*—The condition is not very rare. It may be primary from disease of the blood-vessels or purpura hemorrhagica; or it may be secondary to myelitis and tumors. Primary hemorrhage occurs sometimes in infancy, but usually in males between the twentieth and fortieth years. Infection, injuries, over-exertion and exposure, excessive coitus (Gowers), syphilitic disease of the blood-vessel, and convulsions are causes. The disease sometimes occurs in old people with degenerated arteries, which break and lead to a spinal apoplexy, just as occurs in the brain. More often the condition in old people is a thrombosis. A special cause of spinal hemorrhage is working under high atmospheric pressure, as in the caisson disease.

The *symptoms* develop rapidly, with at first feelings of numbness or weakness for one or two hours or longer. Then there is a sudden paraplegia, with anæsthesia or ataxia or both. The anæsthesia is often dissociated, there being loss of pain and thermic sense with retention of considerable tactile sense. The sphincters may be paralyzed; the urine has to be drawn. The reflexes may be abolished at first, but soon return and become exaggerated with signs of pyramidal tract involvement. There is considerable pain in the back. If the lesion is high up, the arms and thorax are involved. The acute symptoms begin usually to subside at the end of seven to ten days and the disease takes the character of a chronic myelitis. If improvement does not occur, evidences of acute myelitis or softening appear and the patient dies.

Pathology.—The vessels involved are the central arteries, which supply the gray matter and are under relatively high pressure. The rupture of the vessel, when due to disease, is caused by a fatty degeneration of the coats or endarteritis; miliary aneurisms, such as are found in the brain, rarely develop in the cord. Hemorrhage often precedes or begins a myelitis, of which it may be the cause or the result. The clot may be absorbed, leaving a cavity as in the brain; or the broken-down tissue may become the centre of a myelitic focus. The hemorrhage is usually single, but there may be several. Multiple capillary hemorrhages

occur, but usually only from asphyxia and convulsions or in caisson disease. It is possible that some of the cases of disseminated myelitis occurring after infectious fevers start from small hemorrhages. Hemorrhage sometimes results from the invasion of the cord by a new growth, as in syringomyelia.

Diagnosis.—The points to be noted are the sudden onset without long premonitory symptoms, and the absence of fever followed later by gradual improvement. There is much less pain and more paralysis than in meningeal hemorrhage, and the dissociation of cutaneous sensations is very characteristic. In acute softening there is less of the dissociation of sensations and usually a more extensive paralysis and the patient is syphilitic or aged. The disease is often mistaken for acute primary myelitis, which does in fact sometimes follow it.

Prognosis.—This is often serious as regards life, and always serious as regards health. It depends on the extent and seat of the hemorrhages. Dorsal hemorrhages are more favorable, cervical the least.

Treatment.—Absolute rest, ice bags to the spine, and small doses of aconite given early are all that can be tried, except the use of symptomatic remedies. Treatment must be applied at once. The late treatment is the same as that for chronic myelitis.

THE CAISSON DISEASE (DIVER'S PARALYSIS)

The caisson disease is the name given to a group of symptoms characterized mainly by pains and paralysis which occur in persons who work in caissons or diving bells, and which are brought about by the sudden return from condensed air to the normal atmosphere.

Etiology.—Persons employed in caissons or bells work usually under a pressure of from one to four atmospheres, which means a pressure of from fifteen to fifty pounds to the square inch. Accidents, rarely, if ever, occur if the pressure is not over one atmosphere, and they are rare if the person has not been subjected to the pressure for at least an hour. The effective cause of the symptoms, however, is a too rapid decompression. Different persons vary in susceptibility to the effects of this change in the atmospheric pressure, and those unused to the work or of maturer age and poor arteries are more liable to be attacked. Naturally, the disease is seen only in men, and during the working period of life. Hunger and exhaustion make a person more susceptible.

The symptoms set in usually very soon after the patient has come out from the caisson, but they may be delayed for half an hour to an hour. They consist of intense neuralgic pains in the lower extremities, often affecting especially the joints. There is at the same time epigastric pain. Nausea and vomiting with weakness in the lower limbs, amounting in some cases to absolute paralysis, very soon appear. There may be

headache, dizziness, and sometimes choking, coughing and oppression in breathing. If the paralysis is considerable, it is usually accompanied by anæsthesia. Disturbances in the sphincters, with retention of urine and constipation, may also be present. The symptoms vary very much in severity, from pain, weakness in the legs and nausea, up to frightful neuralgic attacks and complete paralysis, motor and sensory. The upper limbs are rarely affected. The disease lasts from a few hours up to several weeks. Death occurs in some of the very severe cases. The symptoms having reached their climax gradually ameliorate, and a complete cure is not infrequent. In some instances, however, the patient is left with a permanent paraplegia and the ordinary symptoms of a

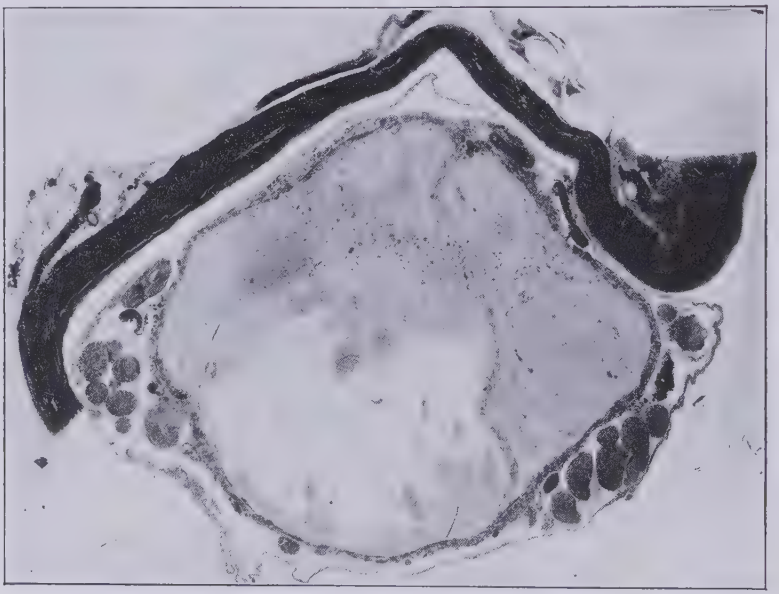


Fig. 98.—Spinal cord in caisson disease showing extensive destruction of tissue.

transverse myelitis. Hemiplegia occurs in about one-fifth of cases and still more rarely a monoplegia.

The disease in its mildest form is characterized mainly by severe pains, with some weakness and dizziness which usually soon pass away. The symptoms are spoken of as "the bends." When vertigo and staggering and some mental confusion are dominant, it is called "the staggers," and the dyspnœic and coughing attacks are called "the chokes."

Pathology.—When the patient is under atmospheric pressure in the caisson, the blood is driven from the surface of the body, and the internal viscera, including the brain and cord, are congested. The sudden change from the abnormal to normal pressure produces a rapid flow of blood from

the internal organs to the periphery. The viscera not inclosed in bony cavities are enabled to relieve themselves of this congestion without much harm, but the circulation in the brain and spinal cord is less elastic; that in the spinal cord being less even than that in the brain. The result is that congestions and small hemorrhages ensue, producing a destruction of the nerve-tissue. In other cases there seems to be a blocking up of some of the small vessels, with consequent softening of different portions of the cord and to a less extent of the brain. The most important element in causing the trouble is the escape of oxygen and carbonic-acid gas from the blood into the tissues or into the blood-vessels, forming gas emboli. It will be seen, however, that on the whole the serious organic changes consist in the vascular disturbances with rupture or obliteration of blood-vessels, and consequent destruction and necrosis of tissue. Following this is a reactive inflammation producing the phenomena of a reactive acute myelitis.

Prognosis.—The painful and vertiginous types of the trouble nearly all get well. Over half of the paralytic cases recover.

Prophylaxis.—The safest age for caisson workers is from twenty to thirty-five. They should have a good muscular system, good eyes, ears, lungs and hearts. The Anglo-Saxons and negroes seem to stand the work better than the Latin races (Pelton). They should not have the status lymphaticus. They should drink but little. The most important precaution of all is slow decompression, about two pounds per minute. Hill and Greenwood assert that pressures up to seven atmospheres (about 140 pounds to the square inch) can be safely borne, if the decompression is slow enough. On coming out the workmen should not do any climbing, and should not take hot coffee, nor alcohol, as a drink.

Treatment.—The most essential thing for immediate treatment and relief is immediate recompression in a hospital-lock provided for the purpose. The pressure (Pelton) should be gradually increased to about that under which the patient has worked. Then in a few minutes decompression is very slowly given.

When the disease has developed it can be treated only by symptomatic remedies. The patient should be kept quiet, and given, if necessary, hypodermatics of morphine. Dr. A. H. Smith recommends the use of ergot. Later on, the various neuralgic and paralytic symptoms may be treated on the same principles as those employed in myelitis.

INFLAMMATION OF THE SPINAL MEMBRANES (SPINAL MENINGITIS)

The meningeal inflammations acute and chronic are:

Pachymeningitis.	Affecting the dura mater.
Leptomeningitis.	Affecting the pia mater.
Meningo-myelitis.	Affecting both membranes and cord.

EXTERNAL MENINGITIS, PACHYMENINGITIS EXTERNA

Etiology.—The disease is rare, and always occurs secondarily to some other morbid process. This process is in most cases tuberculosis causing caries of the vertebræ. Other causes are suppurative inflammation, such as a carbuncle, in the neighborhood of the vertebræ, psoas abscess, purulent pleurisy, and the extension of a neoplasm. Tuberculous pachymeningitis is so commonly associated with cord changes that it is described more fully under the head of Compression Myelitis.

Symptoms.—The symptoms are those of irritation of the motor and sensory roots; later of compression of them and of the spinal cord. There occur local pains in the back, radiating pains, local tenderness, and hyperæsthesia, twitching of muscles and rigidity of spine, progressive development of a spastic paraplegia, exaggeration of reflexes, and involvement of the sphincters. Anæsthesia occurs in severe forms, which takes the distribution of a root anæsthesia.

The disease, when chronic, may extend to the other membranes and cord, causing chronic meningo-myelitis.

Pathological Anatomy.—The inflammation, if acute, is generally a fibro-purulent one. The dura mater is covered by a layer of caseous, semisolid matter, often very thick and most extensive posteriorly. It involves the dura vertically for several inches. In chronic forms the deposit is made up of connective tissue and the cord is compressed. In purely suppurative forms the cellular tissue outside the dura is infiltrated with pus throughout a great part of the canal.

The *diagnosis* is based on the presence of the primary local disease, a kyphosis, the radiating pains, and tenderness and by the combination of motor and sensory irritation and paralysis. Anæsthesia and sphincter troubles come late in the disease.

The *prognosis* is bad, if the original disease is a serious one. Still, surprisingly good results are often obtained when the disease is taken early, especially in tuberculosis cases.

The *treatment* consists in attention to the local caries or inflammatory focus. It is therefore purely surgical, mechanical and symptomatic.

PACHYMENINGITIS INTERNA

(*Hypertrophic Cervical Pachymeningitis*)

Inflammation of the inner surface of the dura mater has been much written about, and but little seen. It is almost always only a syphilitic meningo-myelitis and it attacks the cervical region as a rule.

Etiology.—The disease occurs always in adult life. It usually affects males. Syphilis and trauma are the causes.

Symptoms.—The disease begins gradually with symptoms of irritation (irritative stage). The patient suffers from pain and stiffness in the neck. The pains radiate up to the occiput and down the back; numbness, prickling and pain are felt in the arms, more in one than the other. The pains exacerbate and are worse at night. Stiffness and cramps may affect the arms. Nausea and vomiting sometimes occur.

After five or six months, symptoms of paralysis appear (paralytic stage). The arms are affected. They become weak, atrophy occurs, associated with contractures and rigidity. There is still pain, and in addition anæsthesia, hyperæsthesia and trophic changes occur. Later, paraplegia, with rigidity, exaggerated reflexes, and spinal trepidation develop. The patient becomes weaker, and finally dies of exhaustion or from some intercurrent trouble. Usually it runs a long and painful course, but if early recognized and treated it should be at least controlled.

Pathology.—The disease starts as an inflammatory syphilitic exudate upon the surface of the dura. This leads to a chronic inflammatory process, and finally the cord is encircled and compressed by a dense laminated connective-tissue mass, which involves the pia and to some extent the cord substance.

Diagnosis.—This must be made from tumor, myelitis, Pott's disease, wry-neck and progressive muscular atrophy. The history of injury, the slow progressive course, and the localization of the symptoms, their bilateral character and the pain, a history and laboratory evidence by lumbar puncture of syphilitic infection give adequate help. In spinal tumor the symptoms at the beginning are more sharply localized. They develop more rapidly and the course of the disease is shorter than in meningitis.

Treatment.—The syphilitic origin of the disease must be borne in mind. Counterirritants, electricity, hydrotherapy and symptomatic remedies for the pain and spasms are indicated.

ACUTE SPINAL LEPTOMENINGITIS

(*Inflammation of the Pia Mater of the Spinal Cord*)

Etiology.—This is so rare a disease, occurring alone, that I leave it out of the category of independent disorders. The description of cerebrospinal meningitis covers sufficiently the ground.

CHRONIC LEPTOMENINGITIS (NON-LUETIC)

Etiology.—This disease, which used to be often diagnosticated, is now believed to be rare and always secondary to trauma, or an acute process, such as a cerebrospinal meningitis. It occurs oftenest in adults and in

males. Trauma, and especially concussion of the spine, used to be thought a frequent cause, but in most of such cases the trouble is simply a hyperæmia or else is neuralgic and functional.

The *symptoms* gradually develop after an acute meningitis or an injury, they are similar in character to those of the acute process. There are pain in the back, increased on movement and radiating about the trunk and down the limbs, tenderness along the spine, stiffness of the back, twitching and spasms in the limbs with some weakness. The symptoms run an irregular course with periods of improvement.

Pathological Anatomy.—The inflammation consists of a proliferation of connective tissue (productive inflammation of Delafield). The result is a thickening and opacity of the pia mater and arachnoid.

The *diagnosis* must be made from functional disease, meningo-vascular syphilis, myelitis, and vertebral caries.

In vertebral caries the pain and tenderness are much more localized, and there is spasmodic fixation of the trunk. The pain is more continuous and dull, and is increased by lateral pressure and lessened by extension. There is usually also some deformity. If compression occurs there is exaggeration of the reflexes and paraplegia without much anæsthesia.

Treatment.—Chronic meningitis not the product of syphilis is the relic of traumatism or of an acute process and the indications for treatment are simple. Rest is the essential thing. With this can be combined the systematic and persistent use of counterirritants. The hot iron is usually best, because its wounds heal so quickly. Cupping is also useful if done vigorously and often.

MYELITIS AND MYELOMALACIA—INFLAMMATION AND SOFTENING OF THE SPINAL CORD

Inflammation of the spinal cord is known as myelitis. Softening of the cord is known as myelomalacia. The processes are practically associated. Acute myelitis always ends in some form of softening; and acute softening is often followed by reactive or infective inflammation. If inflammation affects the gray matter only it is called poliomyelitis; and if the anterior horns chiefly, it is an anterior poliomyelitis. Both the gray and white matter are usually affected, and myelitis or spinal softening may be diffuse, disseminated, or transverse in accordance with the distribution of the process. The forms are still further divided, in accordance with their location, into the cervical, dorsal, and lumbar. Myelitis is given different names also in accordance with its cause. Thus we have hemorrhagic myelitis, a form in which the process is due to or associated with a hemorrhage; compression myelitis, due to vertebral caries and the pressure of tuberculous exudate, purulent myelitis or abscess of the cord; and

tuberculous and syphilitic myelitis which are really essentially necrotic and softening processes and not myelitic.

The following table will show the different morbid processes and the six clinical types of myelitis:

FORMS OF MYELITIS AND MYELOMALACIA

Pathological Process	Clinical Type
An exudative, lymphogenous inflammation due to infection.	1. Anterior poliomyelitis, Landry's acute ascending paralysis.
An exudative, hemorrhagic or purulent inflammation due to infection.	2. Acute transverse, diffuse and disseminated myelitis. 3. Abscess of cord (rare).
A subacute degenerative and necrotic process due to toxins.	4. Subacute combined and diffuse cord degenerations (combined sclerosis).
An embolic or thrombotic process.	5. Acute and subacute softening of cord.
A parenchymatous degeneration with connective-tissue proliferation due to vascular and toxic causes, or secondary processes.	6. Subacute and chronic poliomyelitis.

Acute myelitis is a local or diffuse exudative inflammation with more or less necrosis and softening. (Type 2 of table.)

Etiology.—It affects adults in early and middle life, and men oftener than women. It is rarely seen in old age or childhood. Heredity and neuropathic constitution are not important factors, though it is probable that there are some persons whose spinal cords are less resistant to infection than others.

Exposure to cold, bodily and sexual excesses, and violent concussion have some importance indirectly in favoring the entrance of infection.

The list of local bodily infections which are the source from which the cord becomes infected is very long and includes general sepsis, peritonitis, appendicitis, enteritis, colitis, liver abscess, cystitis (urinary paraplegia), gonorrhœa, osteomyelitis, and tonsillitis. It more rarely follows general infections, such as influenza, small-pox, scarlet fever, typhoid fever, rheumatism, diphtheria, and pneumonia. Sometimes the micro-organism of acute poliomyelitis causes a definite transverse or diffuse myelitis. Myelitis has occurred associated with the toxæmia of pregnancy. In this and perhaps other infections or toxæmias the lesion may be due to thromboses from the increased coagulability of the blood or circulatory

weakness. The infection of polyneuritis in rare cases may affect the cord also.

Certain chemical poisons such as carbonic oxide, sulphide of carbon, and chloroform cause a necrotic and degenerative rather than an exudative myelitis.

Syphilis does not cause acute myelitis but only softening with hemorrhages and chronic degenerative changes.

The mechanical injury due to gas bubbles in caisson disease may destroy cord tissue and lead to inflammatory changes.

Despite the above long list, acute myelitis is really a very rare disease.

Symptoms.—The initial symptoms consist of feelings of numbness, usually in the feet and legs, which seem heavy and weak. Some pain may be felt about the back. The patient finds that he cannot walk easily, that he moves his legs with an effort and that they feel stiff. In one or two days some paraplegia with anæsthesia has developed, and if the lesion is in the cervical cord the arms are paralyzed also. Partial or complete retention or incontinence of urine and constipation occur at the same time. There may be some fever.

In three or four days, sometimes in a day, the disease reaches nearly its height and the patient is paraplegic and confined to bed.

If the patient is now examined it will be found that he cannot walk or stand, but can move his legs a little. He complains of a sensation like a band around his waist or at the level of the spinal lesion (girdle symptom). His legs feel numb and heavy, but there is little pain and no tenderness. Anæsthesia to touch, pain, and temperature and of deep sensibility exists, in varying degree according to the seriousness of the case, as high up as the lesion. The anæsthesia, if not total, is greatest to touch, next to temperature and to pain and least to sense of position and deep pain. The bladder is anæsthetic; the urine is retained, and has to be drawn. The bowels are constipated, and if enemata are given the fæces may pass away without his knowledge, owing to rectal anæsthesia. If the lesion is lumbar, there is abolition of the sexual power; but if dorsal or cervical, erections may occur without the patient's feeling them. When the lesion is above the lumbar cord also, the bladder may automatically and forcibly contract and expel the urine. In time the bowels regain some power. The paralysis in the limbs affects the extensors more than the flexors of the toes, the flexors of the feet and legs more than the extensors. The patient can push down his limbs better than he can draw them up and he can adduct better than abduct.

The temperature of the limbs for a few days may be raised, but after this it may fall a few degrees below normal. The skin becomes rough, cold, congested; or excessive perspiration may take place. The general bodily temperature is usually normal throughout the disease, but in

some cases septic fever develops of 102° to 104° and continues. The prognosis is then bad.

Bed-sores may develop early, within a few days or weeks unless great care be taken. They appear oftenest upon the buttocks and heels, and are due to vascular weakness trophic disturbance, combined with pressure and pyogenic infection of the parts. Bed-sores are, however, not necessary if every possible care and precaution are taken.

If the lesion is lumbar, the tendon and skin reflexes are lessened and the paralysis is somewhat flaccid. The muscles also tend to waste and show degenerative reactions. If the lesion is dorsal, as is more often the case, the reflexes are present, and after a time become exaggerated;

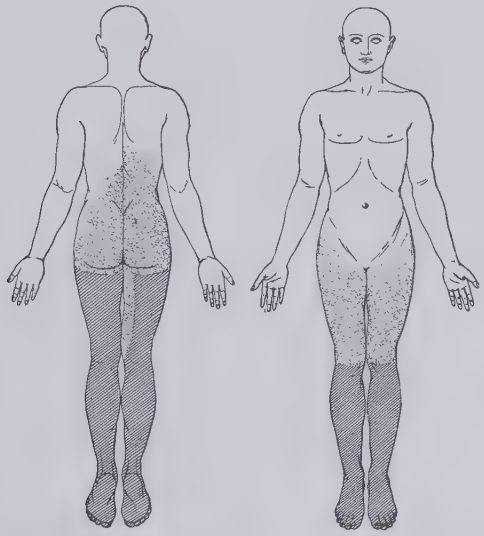


Fig. 99.—Acute transverse myelitis of lumbar cord, showing distribution of anæsthesia. Area in lines = total anæsthesia and analgesia. Dotted area = analgesia only.

there is ankle clonus and extensor response on irritating the soles of the feet; contractures and spasms develop; the legs become drawn up and deformities are produced. If the lesion is so complete as entirely to cut across the cord, there may still be some excessive muscular tension, but the reflexes will be abolished. When the cervical region is attacked, the arms are involved as well as the legs, with a segmental lower motor neuron palsy, generally severer in the arms. There may now be also unequal dilatation of the pupils from involvement of the cilio-spinal centre. In extensive involvement of the upper part of the cord there will be paralysis of the intercostal muscles and disturbance of the heart's action.

The disease, having in a few days reached its height, usually remains stationary for a few weeks, and then, should the patient live, imvproement slowly sets in. In some cases evidences of extension upward or downward occur (ascending or descending myelitis); the symptoms become more severe, and in a few weeks, or oftener months, death occurs directly from bronchopneumonia or pyonephrosis.

As improvement begins, a return of sensation is first noticed (one to six months); this is followed by return of more or less motion (six to eighteen months). Spasms and contractures now develop, owing to secondary degenerations. A certain amount of ataxia from posterior column degeneration, with a little anæsthesia of the skin, may remain, so

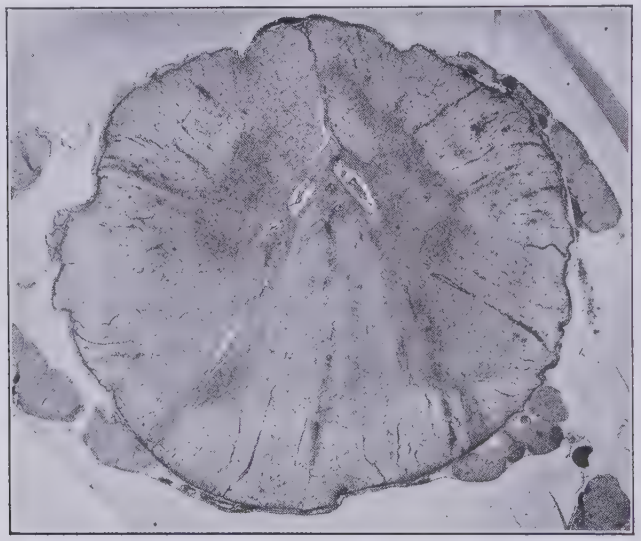


Fig. 100.—Acute diffuse myelitis.

that, if the patient has sufficient motor power to walk somewhat, he presents many features of "ataxic paraplegia."

Some additional and continued improvement may be expected for from one to two years. A few cases get almost entirely well. The majority become more or less paraplegic and bedridden, in which condition they are regarded as cases of chronic myelitis, though no active process exists.

Pathology.—The infection of the spinal cord in acute exudative types of myelitis is believed to be through the lymph paths of the nerves and not through the blood-vessels (lymphogenous myelitis). In the more severe hemorrhagic and in the purulent types it is a vascular infection (hematogenous). The early changes found are those of inflammation,

hemorrhagic extravasation and softening. Often it is impossible to say whether the primary process was inflammatory or due to a hemorrhage or softening.

Macroscopically, the cord at the affected part appears soft, swollen, and either red and hyperæmic or pale and anæmic. In rare cases no change is apparent to the naked eye. In later stages the parts are white or gray, shrunken and hard. The cord may be reduced to a thin shred. The meninges about the affected parts are often thickened, inflamed, and adherent.

Microscopically, if the process is primarily inflammatory we find intense congestion, distended blood-vessels, emigrated white blood-cells

Fig. 101.—Acute hemorrhagic myelitis.

in great number, especially in the perivascular spaces, swollen axis-cylinders and œdematous swelling of the myelin sheath, red blood-cells, cells filled with fat granules known as compound granular corpuscles, or Gluge's corpuscles (Fig. 100).

When the infection is very intense the hemorrhagic process is more active (Fig. 101).

In fatal cases inflammation and softening continue; fresh areas of cord are involved, much meningeal exudation takes place and finally death occurs. The process may in very rare cases be still more acute, suppuration and abscess occur, and here death ensues in two or three weeks.

The inflammatory and softening processes above referred to are described in accordance with their appearance as red softening, yellow softening or white softening. A form of so-called inflammation, known as inflammatory œdema, is also described. It is an abortive inflammation, "a lymphatic congestion," analogous to vascular congestion.

The *diagnosis* of acute myelitis must be made from hemorrhage, non-inflammatory softening, multiple neuritis, and hysterical or functional paralysis. Syphilitic spinal paralysis (Erb's type) is recognized by the cerebrospinal fluid tests. Spinal hemorrhage comes on suddenly and is not attended by fever. If meningeal, it is attended with pain and the condition soon remits. Acute softening not syphilitic usually occurs only in old people with spinal arterial sclerosis. In softening there is no constitutional disturbance, no leukocytosis, the process is slower, there is less pain and the dissociation of cutaneous sensations is less marked. In acute ascending paralysis the disease is progressive, there are no involvement of sensation, no atrophy, and little change in the electric irritability. In multiple neuritis the onset is slower, there are more pain, local tenderness and sensory disturbance, and the sensory loss is of the glove and stocking types; the bladder and rectum are rarely involved. In meningitis there are pain and tenderness in the back and limbs, rigidity, cramps, a little paralysis, and no bladder trouble. In hysterical paraplegia there are no marked atrophic changes, but little spasm or rigidity, no electrical changes, and the stigmata of hysteria may be found. The sensory disturbances are variable and somewhat characteristic (see Hysteria), and the knee-jerks are not greatly if at all exaggerated. Tapping of the spinal canal will enable one to exclude hemorrhage and to determine by the presence of a lymphocytosis that there is an inflammatory process. The serological tests will also determine whether it is a syphilitic process or not.

The diagnosis of the *location of the lesion* is made by studying the height of the anæsthesia, the skin reflexes (see p. 46), the loss of sphincter control, and the distribution and extent of the paralysis. There is often a differentiation of the anæsthesia, as shown in Fig. 99.

Prognosis.—The prognosis is worse the more complete and extensive the paralysis. It is worse in serious motor paralyses than when sensation is chiefly involved. It is best in dorsal myelitis and worse usually in cervical myelitis, other things being equal. Bed-sores and slight fever are unfavorable signs; so also is severe involvement of the bladder and rectum. Recovery of sensation gives good hope of recovery of some motion. Total absence of recovery of sensation and motion after six months is very unfavorable. Improvement may be expected up to eighteen months after

the onset, and in some cases even longer. In compression myelitis there is more chance of recovery than in the other forms provided that the compressing agent be removed.

Treatment.—In the attack, the patient must be put to bed; diaphoresis should be promoted, a laxative and colon washings given, and, in general, eliminative treatment followed. Urotropin and small doses of aconite and nitroglycerin may be prescribed. The bladder should be watched. After a week, moderate doses of iodide of potassium should be given. After about three weeks, if there is no fever, electricity may be applied carefully and strychnine administered in small doses. Bedsores should be guarded against by the use of water-beds, or cushions, absorbent cotton, bathing the parts with alcohol and weak solutions of tannin. Infusion of buchu, boric acid and tincture of hyoscyamus will often help the bladder disturbance. Urotropin and sodium benzoate may be also needed. The frequency of syphilis as a cause of acute paraplegia (nearly one-half the cases) should lead to the persistent use of medication for this condition if indicated. After acute symptoms subside, the treatment is that for chronic myelitis.

CHRONIC MYELITIS AND SOFTENING

(*Transverse and Compression Myelitis, Erb's Syphilitic Spinal Paralysis*)

Chronic myelitis is the name given to a disease characterized by a chronic inflammation of the spinal cord and to the chronic reparative processes which follow acute inflammation, injury and softening. Chronic myelitis is usually a mixture of inflammatory, reparative and necrotic processes and may be only the terminal stage of acute inflammation or softening.

Forms.—Different names are given to chronic myelitis in accordance with the part of the cord affected. Usually the disease affects only certain levels, and then it is called *transverse myelitis.* More rarely it is *diffuse* or *disseminated, central* or *marginal.* When caused by pressure from vertebral disease, it is called *compression myelitis,* though this is really often only an inflammatory œdema secondary to a peri-pachymeningitis.

Etiology.—The disease may be either *primary* or *secondary.* The *primary* form is somewhat the rarer. It occurs chiefly in adults and in early and middle life, and much oftener in males, and it is due to syphilis. *Secondary* chronic myelitis is a rather common form. It is really only the later stage of acute myelitis, softening, destructive hemorrhages and injuries.

Meningitis may extend and cause a meningomyelitis. A neuritis may possibly ascend and cause myelitis, but such cases, if they occur are very rare. When all is said, chronic myelitis as usually seen is a syphilitic myelitis, or the result of injury, of hemorrhage, or of vertebral caries.

Among 67 personal cases of secondary chronic myelitis there were 61 men and 6 women. The causes that can be assigned were: Syphilis, 23; injuries, 12; exposure, 3; acute infection, 6, of which 3 followed grip, 1 typhoid, 1 meningitis, and 1 mumps. Two were due to caisson disease; 4 were of arteriosclerotic origin occurring in the aged and 6 were due to tuberculosis. A study of the age shows that practically all the cases occur between the ages of twenty-one and fifty, and most of them between the ages of thirty-one and forty. Those cases occurring in the extreme of life are due to senile arterial changes or injury.

Symptoms of Primary Chronic Myelitis.—When the disease begins primarily as a chronic affection the symptoms are as given below. And since nearly all such cases are of syphilitic origin, the description of primary chronic myelitis is practically that of *syphilitic spinal paralysis* (Erb's *spinal paralysis*).

The patient notices that his legs seem heavy and easily get tired; prickling and numb sensations are felt in the feet; occasionally a little pain develops in the back or there is a sense of constriction about the trunk. The legs are stiff, and tests often show that the reflexes are exaggerated, with ankle clonus and extensor response (sign of Babinski). There is but little wasting of them, however. The sexual power declines; the bladder gives some trouble, there being a tendency to retention; the bowels are constipated. After a few weeks or months there is a partial paraplegia, with rigidity of the limbs and exaggerated reflexes. Some anæsthesia exists, and occasional pain, which is not severe and is felt more in the back than the legs. Sensory loss is usually slight compared to motor loss.

The muscles have later wasted somewhat, but show no decided changes to the electrical current. The bladder becomes more involved, the urine has to be drawn, it is often alkaline, and unless care is taken cystitis develops. The patient is still able to walk, but he does so with a stiff, shuffling gait which is characteristic (Fig. 102). The disease may show signs of slowly extending up and down, more often up. The arms become involved; weakness and stiffness, with some wasting, anæsthesia and pain, develop, or the disease may cease its progress and the patient remain partly paralyzed for years. The general health during the course of the disease deteriorates slowly; the patients often become anæmic and have an unhealthy pallor. Eventually the paraplegia may become complete,

the patient is bedridden, the legs are atrophied, contractured and rigid, with more or less anæsthesia. Cystitis and nephritis develop, and the patient dies from these or other intercurrent diseases.

Chronic secondary myelitis, which is the form often seen, presents eventually much the same picture as that just described. In this type, however, the symptoms are those of an acute myelitis first, then improve, then become stationary, and finally grow worse.

Chronic compression myelitis, so called, is usually only a compression œdema with partial atrophy. It is due, as a rule, to vertebral caries and pachymeningitis, but its cause may be a spinal tumor or aneurism. Compression myelitis is distinguished from other forms by its slow onset and the presence at first of irritative or "root" symptoms. The patient suffers from pain and tenderness localized at a certain point in the spine. The pain radiates about the trunk or down the limbs and is increased on movements. At about the same time some motor weakness develops, usually in the form of paraplegia, one leg being often affected some weeks or months before the other. The muscles waste but slightly. The reflexes are exaggerated; twitchings, spasms and contractures finally occur, and there is developed a spastic paraplegia or quadruplegia. With this there is usually some anæsthesia, though it is not complete. The disease is oftenest in the dorsal or lower cervical region, and hence the sphincters escape until late. Locally, evidences of spine disease may appear early in the form of a kyphosis.

FIG. 102.—Chronic myelitis.

A *central or peri-ependymal myelitis* can rarely be recognized with certainty. It produces less pain and irritation, but leads to muscular atrophy, disorders of sensations, such as thermo-anæsthesia, disturbance of vasomotor and secretory nerves and visceral centres.

Pathology.—The pia mater is thickened over the affected region and often throughout the cord. The cord itself has a gray, discolored look at the affected level, and is usually shrunken or distorted and hard to the touch. In severe cases of secondary character it is reduced to a small size, and the membranes about it are thick and inflamed. In transverse myelitis a vertical area of only two or three inches is involved. The microscope shows that the prominent changes are loss of nerve structure, great increase of connective tissue and increase in the number

of vessels, which often have thickened walls. In the more seriously diseased part little is seen but connective tissue. In parts less diseased some nerve-fibres are seen, many having evidences of partial disintegration. There is also a good deal of amorphous material studded with nuclei. Stellate cells, granule cells, and nerve-cells in various stages of degeneration are present. In the parts less affected the signs of congestion and vascular irritation are more pronounced. Secondary degenerations occur above and below the seat of the myelitis (Figs. 113 and 114). In the typical syphilitic myelitis the meninges are thickened and there is a predilection for the inflammatory change to affect the lateral columns of the cord (Fig. 103).

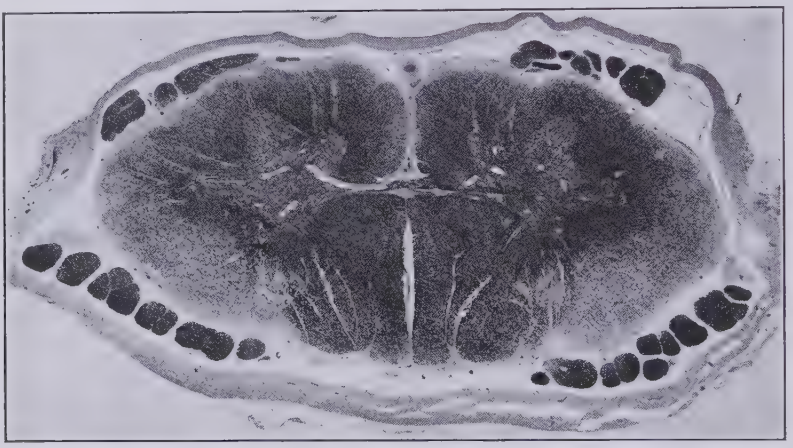


Fig. 103.—Syphilitic meningo-myelitis; cervical region.

Diagnosis.—This must be made from progressive muscular atrophy and amyotrophic lateral sclerosis, pachymeningitis and spinal tumor; from locomotor ataxia, *multiple sclerosis* and brain palsies.

In progressive muscular atrophy there is a peculiar atrophy without involvement of the sphincters or sensory disturbance. Tumors usually cause much more pain; the symptoms come on slowly and are more unilateral at first and more definitely localized. A spastic paraplegia occurs in cerebral diplegia, but the arms are also sometimes involved, and there are no trophic or sensory troubles, nor is there involvement of the sphincters. In locomotor ataxia there is no great degree of motor paralysis, and there are peculiar ataxic and sensory disturbances. In the early stages of multiple sclerosis of the spinal type, however, the symptoms resemble closely a very slowly developing chronic myelitis. The diagnosis can be made by remembering that in multiple sclerosis patient is usually young, that there is absence of much sensory trouble, and

presence upon close examination of eye trouble, tremor, speech disturbance and paræsthesias in multiple sclerosis the course is often a remitting one, the abdominal reflexes are usually lost and the bladder becomes affected quite early. Paralysis from brain disease is almost always unilateral, painless, spastic and free from disturbance of the visceral centres.

Prognosis.—Inflammatory processes have a tendency to cease when their reparatory and eliminative work is done. And this is true of myelitis. However, in the spinal cord secondary degenerations set in as soon as certain tracts are interfered with. Hence chronic myelitis, after a period of improvement, generally progresses, and the prognosis is not very favorable. Still, patients may live comfortably from five to twenty-five years. Dorsal myelitis is the most favorable form; compression myelitis from caries can also often be successfully treated. Syphilitic myelitis and meningomyelitis unless treated are liable to take a progressive course, like a locomotor ataxia. Serious involvement of the bladder is a bad sign, and naturally the prognosis is worse the more complete the paralysis.

Treatment.—In the treatment of chronic myelitis the cause must be sought and treatment applied accordingly. If it is syphilis a thoroughgoing course of anti-syphilitic measures should be instituted.

If it is a compression myelitis from Pott's disease, prolonged rest in bed for six months or more is usually adequate. Jackets and various forms of support are helpful but alone are not enough.

If the cause is caisson disease, injury by fracture or wounds, rest and mechanical measures are the essential things.

Various supplementary and symptomatic measures are to be used. Counterirritation in the form of fly-blisters, or the cautery, may be applied and wet or dry cups used. The descending galvanic current along the spine should be tried; faradism and massage being used upon the limbs. Lukewarm baths, 90° to 98°F., or half-baths with frictoin at 70° to 80°F., are likely to be useful, but these must be tried cautiously. The first baths should last not over five minutes and should be repeated only three or four times weekly. In later paraplegic and bedridden stages, electrical and hydrotherapeutic applications should be followed up patiently and persistently. The patient may be allowed to remain and exercise in the lukewarm bath for some time. The patient may be given courses of tonic treatment, using phosphates, iron, arsenic, and small doses of strychnine. The more surely the disease is non-luetic, the more are simple tonic and mechanical measures alone indicated. For the bladder troubles, the internal use of boric acid, belladonna, ergot buchu, and urotropin are helpful. Salvarsan is of little or no value in long-standing chronic myelitis, unless the disease is progressing.

ACUTE POLIOMYELITIS

(Epidemic Poliomyelitis; Infantile Paralysis)

This disease results from an infection of the body by a specific organism, which produces acute constitutional symptoms of a reactive character; this organism has an especial affinity for the central nervous system, whereby it usually produces an acute flaccid motor paralysis, in muscles segmentally grouped which subsequently undergo more or less atrophy.

Etiology.—It occurs at all ages, but 60 per cent. of the cases are in children under three years. About 15 per cent. of the cases are in adults usually under thirty.

The disease occurs epidemically and to a lesser extent sporadically. Almost all the cases appear in the summer months, especially in August and September. Epidemics occur rather more often on the coast board and in low-lying places; the principal epidemics described have been in Scandanavia from 1903 to 1906, Minnesota, New York 1907 and 1908, Victoria, Australia 1907, Massachusetts 1908, Vienna 1908. Smaller epidemics have been reported from various parts of France, England and the United States. No specific cause can be cited as rendering the body liable to attack; all factors lowering vital resistance are probably contributory to infection, such as malnutrition, fatigue, and other infectious diseases. The incubation period varies, but averages ten days in length.

Bacteriology.—In the summer of 1909 Landsteiner and Popper succeeded in producing the disease in two monkeys by intra-peritoneal injection of emulsified spinal cord obtained from a fatal case of poliomyelitis. They failed, however, to transmit the disease from these to other monkeys. In September of the same year Lewis and Flexner working at the Rockefeller Institute also succeeded in producing the disease in monkeys by means of intra-cerebral injection of poliomyelitic cord. The cords of the affected monkeys furnished a virus which by September 1910 had been propagated through twenty-five separate series of monkeys. The virus is found to pass readily through a Berkefeld or Chamberland filter. It withstands glycerinization, drying and freezing over prolonged periods, but is readily destroyed by heat and by comparatively weak antiseptics such as menthol and a 1 per cent. solution of hydrogen peroxide. In 1913 the virus was cultivated with difficulty by Flexner and Noguchi under anaerobic conditions; subcultures in the fifth generation have successfully reproduced the disease in monkeys. The minute colonies are composed of globular bodies averaging 0.15 to 0.3 micron in size; these occur in short chains, in pairs, and in masses, and are stainable by Giemsa's or Gram's methods. The virus can be inoculated with success into the brain; into the subdural space; subcutaneously; by introduction into the

stomach and into the intestines. It has been shown that it can be carried from the ill to the well through the intermediate agencies of the house fly, the stable fly, *Stomoxys calcitrans*, and the bedbug. Experiments directed to prove an identical rôle on the part of mosquitoes and lice were negative. Flexner and Amoss, however, in papers published in 1914 believe that infection is local and by way of the lymphatics, and not general and by way of the blood. They believe the virus enters the body through the nasopharyngeal mucous membrane, and hence through the lymphatics of the olfactory nerve to the leptomeninges. An attack of poliomyelitis confers an active immunity against a second infection. This obtains apparently for at least three years. Successful vaccination against the virus has been achieved in the *macacus* but not as yet in man. It has been shown experimentally that the previous administration of

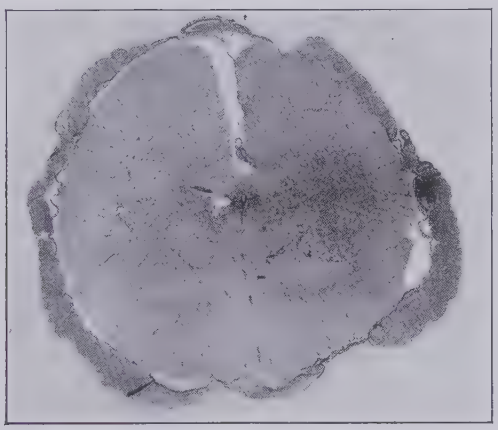


FIG. 104.—Acute anterior poliomyelitis showing diffusely inflamed area on right side.

urotropin will in the monkey increase the incubation period of the inoculated disease and diminish its severity.

Pathology.—Naked-eye appearances: The brain may be œdematous and show some vascular engorgement. The cord also may be œdematous with some wrinkling of the swollen and congested meninges. On section the distinction between gray and white matter may be diminished through softening especially in the severely affected areas, usually the cervical and lumbar enlargements. Similar softenings may be found in the brain stem or hemispheres. The spleen is enlarged and changes varying from coagulation to hemorrhage may be found in the kidneys and liver. The microscopic lesions in the brains and cords of fatal cases consist mainly of congestion and hemorrhage into the gray matter; the anterior horns being disproportionately affected. The entire spinal cord may be involved in a series of vascular accidents of varying degree. The lesions

consist of a small round-celled infiltration in the perivascular spaces with the development of considerable œdema. Arterial thrombosis is never apparent, and the degeneration and necrosis of cells are invariably produced by gross pressure of inflammatory exudates on the cells and the nutrient blood-vessels. A leptomeningeal infiltration of mononuclear cells is always present and is perivascular in position. The greater affection of the anterior over the posterior horns is explicable by the richness of the vascular supply to the former; a similar consideration serves to explain the apparently disproportionate involvement of the lumbar and cervical enlargements (Figs. 104, 105 and 106).

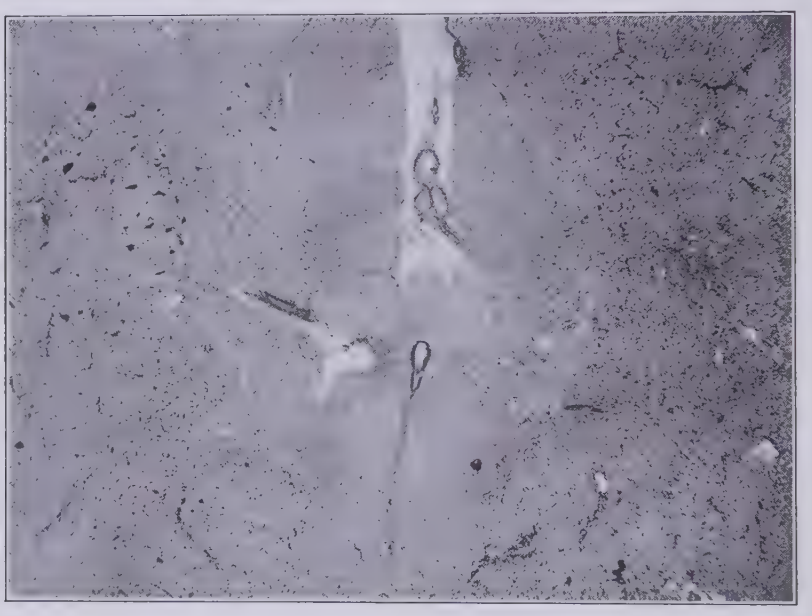


FIG. 105.—Acute anterior poliomyelitis. Same as previous figure enlarged, showing loss of cells in right-horn.

Signs and Symptoms.—These may be conveniently described under different headings: (1) A prodromal period—lasting a few days; (2) an acute stage of a few hours to a week; (3) a stationary period—one to six weeks, usually two weeks; (4) a stage of improvement—six months to three or four years; (5) a chronic stage of residual incurable atrophic palsy.

1. In some cases the onset of the acute stage is so rapid that the prodromal period is practically eliminated, but the vast majority of patients complain of some general malaise, and headache of considerable severity with occasional vomiting. Drowsiness combined with irritability is usually present. There is generalized hyperæsthesia with pain in the back,

in the neck muscles—which may also be rigid, and in the limbs; the pain is usually greatest in limbs subsequently paralyzed. Constipation is usual; there are present much generalized sweating and a moderate fever and leukocytosis in blood and spinal fluid.

2. The constitutional symptoms of the prodromal period may be carried on through the few hours or days of the second stage in which motor paralyses appear. These come suddenly and achieve their maximum severity within a few hours. The affected limbs are toneless and tender. Sensory changes are very rare and transient. Sphincter disturbance is usual but quickly passes. The paralysis most often affects both legs, next one leg, next the arms and legs, and after this various

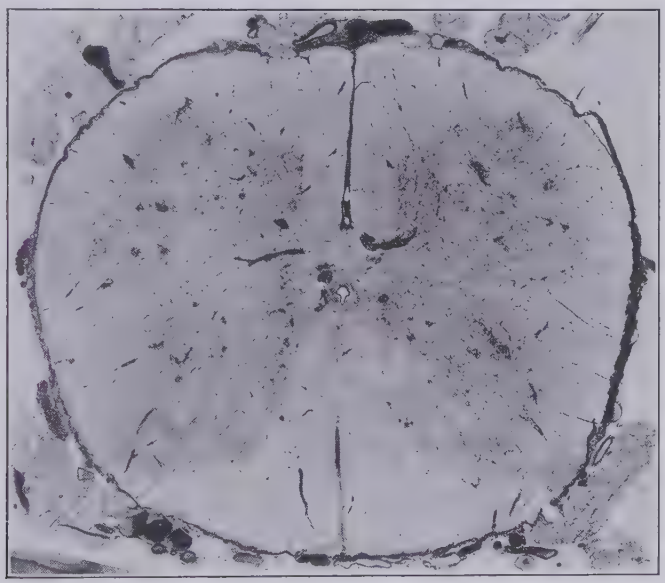


FIG. 106.—Acute anterior poliomyelitis.

combinations. The cranial nerve nuclei may be attacked, and in the not very common cases where the cerebrum is affected there may result hemiplegia of upper motor neurone type with some residual intellectual impairment. The term polioencephalitis superior has been applied to those cases where the oculomotor nuclei are destroyed; affection of the nuclei placed lower in the brain stem has been called polioencephalitis inferior. Cases in this stage prove fatal usually from medullary involvement or paralysis of the respiratory muscles.

3. The paralysis remains at its height for from one to six weeks, and then improvement gradually sets in. In two or three weeks a wasting of the paralyzed limb may be noticed. It is flabby, its temperature is

lowered, and the reflexes may be gone. Slight tenderness may be present but there is no anæsthesia.

4. The stage of improvement lasts for from six months to several years. The paralysis gradually disappears, beginning in the limbs least injured. This continues until but one or two limbs are still affected. In them the muscles are wasted and show the reaction of degeneration, viz., loss of faradic irritability, retention and slowing of galvanic irritability, and sometimes polar changes. In the leg, the anterior tibial and peroneal groups are oftenest affected; in the arm, the muscles of the shoulder girdle. After eighteen months not much further spontaneous improvement can be expected, but much can usually still be done by vigorous massage and suitably adapted exercises.

Fig. 107.—Poliomyelitis, late stage showing atrophied and shortened leg. (*Schænborn and Krieger.*)

5. The temperature of the affected limbs is lowered several degrees; the skin has a reddish-purplish mottled look. The bones as well as the muscles of these limbs do not grow as well as those of the others. Hence in time the extremity is disproportionately small and short (Fig 108). Owing to the contraction of unopposed muscle deformities occur. The most frequent are talipes equinus, talipes varus and valgus. Deformities of the knees, and contraction of the plantar fascia also take place. Spinal curvature is common in cases in which the supporting back muscles have been affected. The general health of the patient is usually good. A consideration of the pathology of this disease will make evident the fact that many different clinical varieties must occur to correspond with the varied combinations of spinal and cerebral lesions produced

The following distinct types may be mentioned:

1. Spinal poliomyelitis—a sudden feverish attack followed by the palsy of one or more limbs.

2. An ascending form in which, first, are involved the legs and later the abdomen and thorax. This type is often fatal.

3. A bulbar type, with palsies of the various pontine and medullary cranial nerve nuclei.

4. Polioencephalitis, from which a state of meningismus with spastic paralyses may ensue.

5. Ataxic polioencephalitis, by which is merely meant a specific inflammatory process, the maximum stress of which falls on the cerebellum. The clinical symptoms are briefly, an acute illness of short duration, followed by motor ataxia of the arms and legs, nystagmus, and explosive syllabic speech.

6. Abortive forms in which only general constitutional disturbances occur, and paralyses are either quite fleeting, or entirely absent. In these, diagnosis may be surmised from the co-existence of an epidemic of the disease, and confirmed by spinal fluid examination. The necessity of making correct diagnoses in these cases is indicated by the fact that poliomyelitis has been propagated through successive series of animals, from the nasal washings of an abortive case.

Diagnosis.—The initial symptoms of poliomyelitis are almost generic for all infected conditions. Influenza must be thought of. Against it one puts the seasonal unlikelihood, it being a winter and spring disease. In poliomyelitis, catarrhal symptoms will be absent. Gastroenteritis is most frequent in the summer and early autumn, but in it the onset is less rapid, pyrexia less sudden, while somnolence, general hyperæsthesia, and sweating are infrequent. Constipation rather than diarrhœa obtains in the early stages of the cord affection. Lumbar puncture should be performed early in all doubtful cases, particularly to differentiate the condition from tuberculous and diplococcal meningitis. The main characteristics of the three fluids may be here contrasted:

Tuberculous meningitis: Clear, forming a fine clot on standing. Globulin content early increased. The normal reduction of Fehling's solution often lost. Usually an almost pure lympocytosis from the very beginning of the disease. In 85 per cent. tubercle bacilli may be demonstrated.

Diplococcal meningitis: Fluid turbid and heavily albuminous. Fehling's test positive except in very chronic cases. Almost pure polymorph leukocytosis. Gram-negative diplococci may be easily demonstrated. (See also "Cerebro-spinal Meningitis".)

Acute poliomyelitis: Fluid clear. Clot formation on standing slight. Globulin content slightly raised in early stages, and gradually rises for about three weeks after which it may sink to normal. Fehling's test normal. In the first three days of the disease there is usually an almost pure polymorph leukocytosis, as high as 1,000 per cubic millimeter. This is then replaced by an almost pure lymphocytosis of gradually diminishing severity. No organisms can be demonstrated by ordinary methods. The disease must also be distinguished from toxic polyneuritis, birth palsies and progressive muscular atrophy. A consideration of the following facts will usually make the diagnosis easy:

1. The age of the patient.

2. The presence of an epidemic of poliomyelitis and its seasonal variations.

3. The abrupt onset and rapid development of extreme paralysis.

4. The tendency to improve.

5. The segmental character of the paralysis; the absence of bladder disturbance and objective sensory losses.

Prognosis.—The patient rarely dies, either from the disease or its sequelæ. In some epidemics, however, there has been a mortality of about 12 per cent. He always improves, but he recovers completely in only a small number of cases. In these, no cell necrosis has occurred but only cell deterioration.

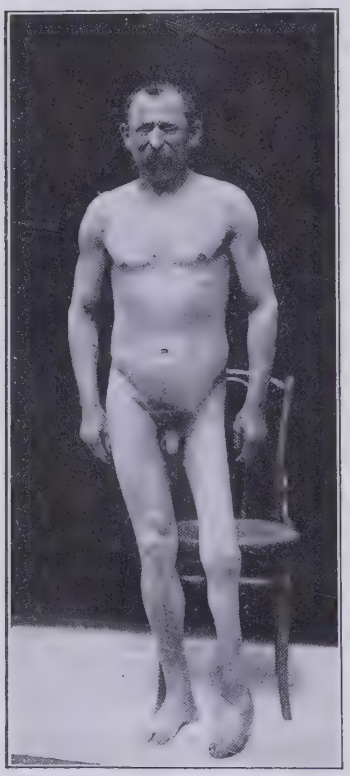


FIG. 108.—Poliomyelitis, late stage, showing atrophy of leg and deformity of foot. (*Schœnborn and Krieger.*)

Treatment.—The patient should be isolated in an airy room, the windows of which are well screened. Fæces, urine and nasopharyngeal discharges must be rendered inocuous by antiseptics as in a case of typhoid fever. A nasal spray of 1 per cent. hydrogen peroxide must be used and maintained for some months. Urotropin must be given immediately and maintained in large doses for at least two weeks. All persons exposed to poliomyelitic infection should be encouraged to use antiseptic nasal sprays and should be given urotropin gr. v. twice daily as a prophylactic measure. Rest is essential. The bowels should be opened and a diuretic given (calomel gr. ii.; tartrat. potas, gr. xx.). Lumbar puncture will aid diagnosis and relieve, at least temporarily, spinal congestion. Sedatives may be required in the early stages. Strychnine is to be avoided. A hot pack twice a day will relieve tenderness. Aconite and sweet spirits of nitre may be given to reduce fever. The limbs should be wrapped in cotton and defended from pressure. When hypersensitiveness has disappeared gentle massage and passive movements should be instituted, for from five to twenty minutes five or six times a day. Electrical applications should be begun cautiously, at first three times a week and, when well borne, daily. Frequent systematic rubbings given by a relative are frequently more productive of good results than are less

frequent treatments given by a trained masseuse. The child must be trained to try to use the limb as much as possible: with this in view it is inadvisable to keep the patient confined to bed after the acute stage has passed: the child should be laid on a well-blanketed floor and encouraged to crawl and push its way about the room. For older children and adults much can be done by the studious performance several times daily of exercises adapted to develop the affected muscles and those adjacent to them. Generally speaking, it is a mistake to employ orthopædic apparatus until two years have passed, nor should they be employed even then unless it be clear that developmental exercises are useless. The same attitude should be adopted toward operations for the correction of deformities or for restitution of function by nerve grafting; such procedures are often of the greatest value but must only be employed after much consideration in selected cases. FOSTER KENNEDY.

CHRONIC ANTERIOR POLIOMYELITIS

(*Remitting Spinal Amyotrophy*)

This disease goes under the name of chronic poliomyelitis, but it shows a closer relation to obliterative vascular disease, than to a simple inflammatory process.

Etiology.—In my experience syphilis has always been present; others give as causes lead, overwork, exposure and trauma. It occurs only in adults and in men more than women.

Symptoms.—The disease comes on subacutely; that is to say, in one or two months. It reaches its height in three or four months and before the end of a year has come to a standstill. The symptoms then usually remain quiescent or improve for one or two years, then take a fresh start and present about the same course, involving new and usually neighboring groups of cells. It may progress no further after this second attack, but there sometimes occur fresh invasions involving finally the bulbar nuclei and leading to a fatal issue. The symptoms of paralysis or weakness precede the atrophy, which is a flaccid one, but in other respects it resembles the ordinary spinal amyotrophies. There are slight or no symptoms of involvement of the pyramidal tracts. There is sometimes slight fibrillary tremor. The disease usually affects the upper extremities and especially the shoulder girdle, but eventually involves the forearms and ascends to the cervical region (Figs. 109, 110).

The tendency to cessation of progress and even to improvement has characterized cases reported by Eisenlohr, and Landouzy and Dejerine. Few cases with autopsy have been reported, and these showed vascular lesions with secondary degeneration of the anterior horn cells and sometimes of the anterior fundamental column.

The Prognosis in the past has been uniformly bad.

Treatment.—The history of syphilis in my cases leads to the recommendation of an anti-syphilitic treatment for this trouble, and this must be very active and persistent.

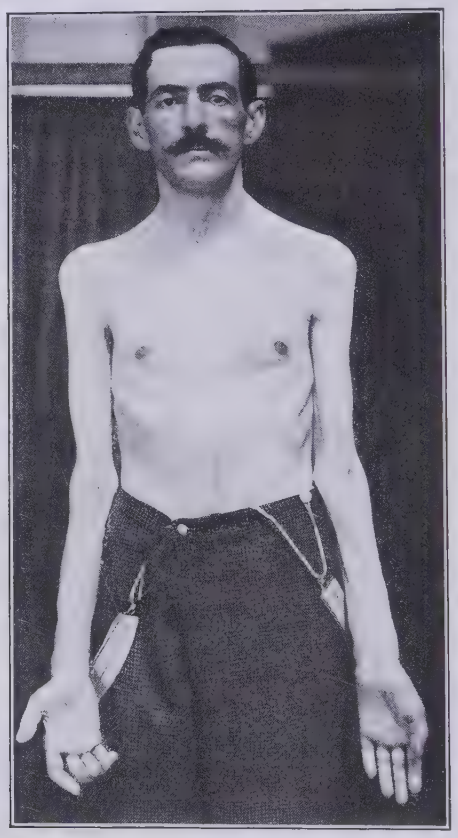


FIG. 109.—Chronic anterior poliomyelitis, luetic right shoulder and left forearm and hand are most affected.

Senile Paraplegia.—Paraplegia sometimes develops in the senile and does so in a somewhat characteristic manner.

In *the spastic form* the paralysis comes on slowly, the legs are stiff, the gait shuffling, the steps very short and the feet are barely raised from the ground. There is some pain in the back, at times, but no marked sensory symptom. There may be decrease in reflexes or no change. The sphincters are not involved. The gait and station suggest paralysis agitans, and some cases may be considered a symptomatic type of this trouble.

A *paraplegia spastica senilis* described by Demange and Oppenheim

is of arteriosclerotic origin and accompanied with signs of pyramidal degeneration.

In *the flaccid form* of senile paraplegia the patient progressively but intermittently grows weaker in the legs, the muscles waste, the gait is feeble, with short steps and great difficulty in going up and down stairs. There is sometimes lumbar pain, and the sphincters may be involved. The patients sometimes improve, but eventually become helpless, though the progress is slow. I have had an autopsy in one case and found very marked arteriosclerotic changes in the anterior horns of the lumbar region with softening. I think that in flaccid senile paraplegias and some of the rigid types there is arterial sclerosis of the anterior vessels with softening and at times small hemorrhages (Fig. 112).

Starr thinks that some forms of progressive senile paraplegia are neuritic and that others are due to a progressive muscular disease (dystrophy).

Fig. 110.—Remitting muscular atrophy. Patient is unable to raise the head.

ACUTE ASCENDING PARALYSIS

(Landry's Paralysis)

Acute ascending paralysis is a disease characterized by a rapidly developing paralysis which begins in the legs and then involves in turn the trunk, arms, respiratory and throat muscles, usually ending in death. There is little disturbance of sensation, no atrophy or changes in electrical irritability, and no involvement of the sphincters. It is thought to be related to acute anterior poliomyelitis.

Wickmann under the name "The Heine-Medin disease" groups together:

1. Acute poliomyelitis.
2. Landry's paralysis.
3. Bulbar and pontine forms.
4. Encephalitic forms.
5. Ataxic forms.
6. Polyneuritic forms.
7. Meningitic forms.
8. Abortive forms.

This assumes that the same infectious or toxic agent may attack the different parts of the nervous system. This is practically the conception taught in the earlier editions of this work. However, all forms of acute ascending paralysis as it is seen clinically are not due to the poliomyelitic organism.

Etiology.—The disease is a rare one. It occurs chiefly between the ages of twenty and forty; men are affected oftener than women. Exposure is an exciting cause, and it occurs sometimes after acute infectious fevers. The form of rabies known as "paralytic" causes a disease which is apparently identical with Landry's paralysis. The form which is caused by the micro-organism of anterior poliomyelitis occurs naturally when epidemics of this disease are present. It is not yet proved that true ascending paralysis is always poliomyelitic.

Symptoms.—There may be slight premonitory symptoms for a few days, consisting of numbness in the extremities, pain in the back or limbs and malaise. The first definite sign of the disease is weakness in the legs, which rapidly increases, until in a day or two the patient cannot walk. The paralysis soon involves the trunk, arms and the muscles of

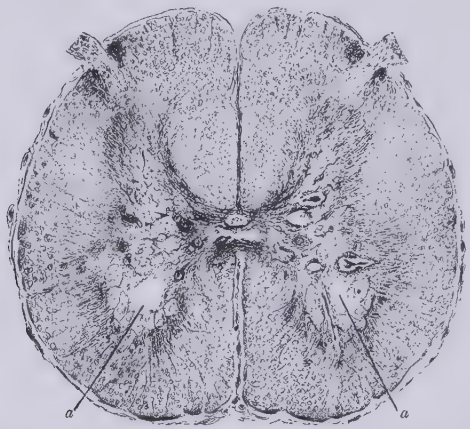


FIG. 111.—Lumbar cord in a case of senile paraplegia, showing atrophy of anterior cornua secondary to vascular disease.

respiration; the medulla is last affected, and then respiration becomes difficult; swallowing and articulation may be impossible. In rare cases there are facial and eye palsies. During the course of the paralysis there is little pain or sensory disturbance, but some degree of anæsthesia may occur. The deep reflexes are abolished. There are no vasomotor and no secretory disturbances, no noticeable atrophy, and no degenerative reactions in the affected muscles. The bladder and rectum are involved only in rare cases. There may be slight initial fever, but none occurs after the disease has well set in. The mind remains clear.

The disease, as a rule, ends fatally, and it usually runs its course in less than a week. Death has occurred in forty-eight hours. On the other hand, death has been postponed three or four weeks.

In other cases the disease stops short of the medulla. The patient

becomes totally or nearly paralyzed below the neck. He then begins slowly to improve, and this improvement continues for one or two years. Eventually a fair degree of health is obtained.

Variations.—The disease has been known to begin in the medulla or cervical region and descend.

Pathological Anatomy.—There are three pathological conditions which may cause the symptoms of acute ascending paralysis: (1) Acute poliomyelitis; (2) acute high transverse myelitis; (3) acute multiple neuritis. These different conditions may coexist at times in the same case. Usually as stated the micro-organisms of epidemic poliomyelitis anterior are the cause. In some cases the symptoms are due to paralytic rabies, in others, the streptococcus, the diplococcus and pneumococcus causing intense diffuse myelitis have been found (Fig. 112).

The prognosis is very grave, but not absolutely bad. If there is reason to suspect the case of being one of paralytic rabies, no hope can be offered.

Diagnosis.—This must be made from the ordinary forms of acute poliomyelitis, acute myelitis, acute multiple neuritis and periodic family paralysis.

Its acute ascending course, absence of fever, of anæsthesia, of atrophy, decubitus, sphincter troubles, and especially the absence of degenerative electrical reactions, of a family history with a history of previous attacks are sufficient to enable one to make the diagnosis. The age of the patient, and the presence or absence of an alcoholic history should be considered. Lumbar puncture should give help. (See anterior poliomyelitis.)

Treatment.—This consists of warm baths or packs, colon washings. and eliminative treatment. Large doses of ergotin, gr. ij., every hour have been successful in one case. Urotropin shonld be given, and lumbar punctures should be made.

CHAPTER XIII

SCLEROSES, DEGENERATIONS, SYPHILIS

Introductory.—The term sclerosis is somewhat misleading. Properly speaking, it is the fibroid (and neuroglia) induration which results from degeneration, destruction or inflammatory irritation. We speak of *degenerative* sclerosis, of an *inflammatory* and of a *neuroglia* sclerosis, or of a sclerosis of mixed origin, according to the nature of the primary disease which caused it. Some writers look upon primary degenerations as parenchymatous inflammations. There is no objection to this point of view as long as we agree upon the essential character of the process. The words "degeneration" and "sclerosis" are often used to indicate the same thing, one being the pathological process, the other the anatomical result. I shall use the term "sclerosis" here in its pathological sense, meaning the process of hardening, in presenting a classification of the degenerations of the spinal cord.

Spinal scleroses.	*a.* Primary and degenerative. Luetic, toxic, connate. (Parenchymatous inflammation)	Posterior spinal sclerosis (locomotor ataxia).
		Lateral sclerosis.
		Combined sclerosis. Hereditary ataxia.
		Progressive muscular atrophy, amyotrophic lateral sclerosis.
	b. Secondary, physiological.	Ascending and descending degenerations.
	c. Inflammatory, infective.	Chronic myelitis and sclerosis following destruction of cord.
		Multiple sclerosis.

The tendency of late years has been to classify diseases of the nervous system on an etiological basis, *e.g.*, as syphilogenous, infectious, tubercular but I believe it still is best to follow the clinical and anatomical methods.

Primary degeneration (parenchymatous inflammation) or primary sclerosis, as one may say for convenience, is a process which begins in the neuron itself and ends in its atrophy, with substitution of neuroglia and connective tissue. As to its *nature*, so far as the microscope shows us, it is a gradual decay and death of the neurons. In some sclerotic processes, like locomotor ataxia, the sclerosis is due to the destruction of the neuron by the toxic activity of the *spirochæta pallida*. The decay is accompanied by the development of irritating products, leucomains toxalbumins, which may produce so active a change in the connective

tissue as to lead to something resembling a secondary or reactive inflammation. This is never of high grade, however, and in some forms of tabes is very slight.

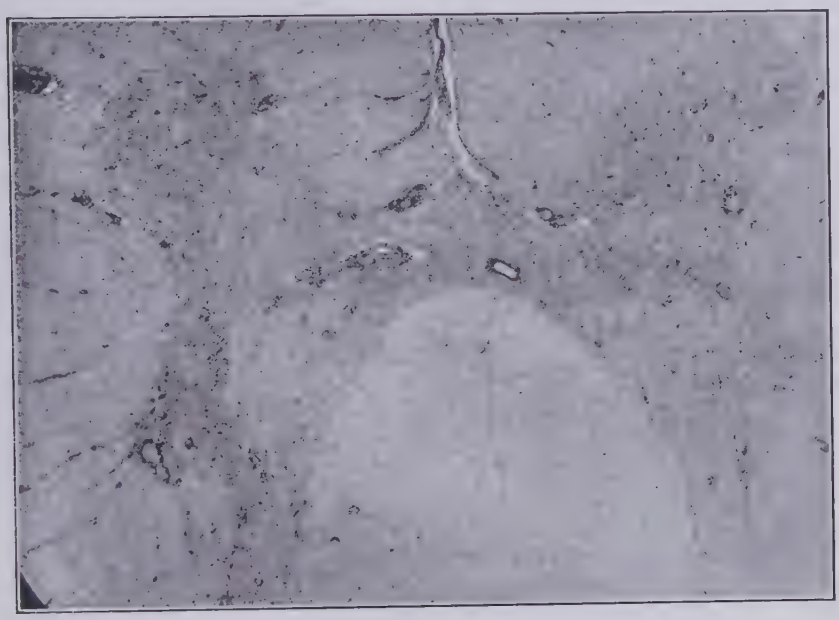


FIG. 112.—Acute ascending myelitis (Landry's paralysis).

FIG. 113.—Ascending degeneration in third cervical segment, from transverse lesion in second dorsal, showing degeneration of columns of Goll and cerebellar tracts.

In progressive muscular atrophy the decay and death produce few irritating products, though enough, perhaps, to account for the fibrillary twitchings and occasional tremulous condition of the muscles.

The ultimate cause of these degenerative processes is the micro-organism of syphilis, toxins, and poisons, and congenital defect in the vitality of the neuron, a condition called by Gowers abiatrophy.

Secondary Degenerations of the Spinal Cord.—When any of the long-fibre tracts of the cord are cut across or destroyed, there soon results a degeneration. This extends up or down in accordance with the direction in which the tracts carry impulses. Thus, when the crossed pyramidal tract is cut across the degeneration extends down; when the column of Goll is involved it extends up. The degenerative process begins almost immediately and is complete in a few weeks. The myelin sheath swells, gradually breaks up and disintegrates; the axis-cylinder is involved next. At the same time the connective tissue and neuroglia proliferate and take the place of the wasted nerves. Finally, long tracts

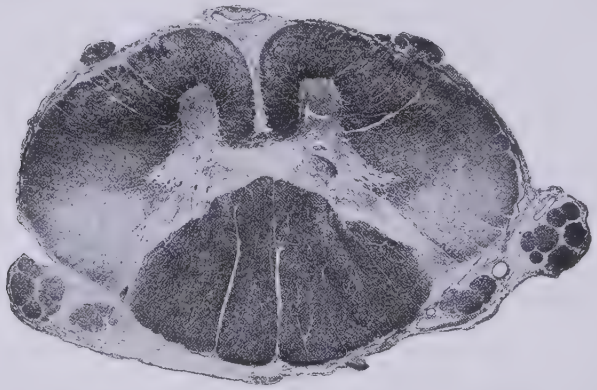


FIG. 114.—Descending degeneration in eighth dorsal segment from lesion in second dorsal, showing degeneration in direct and crossed pyramidal tract, vestibulo-spinal and comma tracts.

of connective tissue have taken the place of the nerve tissue. The process may not be a complete one if the lesion does not entirely destroy the tract.

Secondary degenerations complicate and add to the pathological change in all organic diseases of the cord. In brain disease, involving the motor tract, as in hemiplegia, secondary degeneration extends into the cord and adds to the seriousness of the disease. Degenerations of the spinal cord, however, do not extend up to the brain to any extent, except in the case of disease of the antero-lateral ascending and cerebellar tracts.

Those forms of sclerosis found in chronic myelitis are similar to the connective-tissue scars following destructive inflammation elsewhere. A person who has a chronic myelitis has a cicatrix in his spinal cord.

The sclerosis of multiple sclerosis is probably inflammatory also, but it is a neuroglia rather than a connective-tissue cicatrix.

Short Degenerating Tracts.—By the use of more delicate stains other degenerating tracts have been discovered. These are described under the head of the anatomy of the spinal cord.

The short-fibre tracts degenerate only a little way up and down.

SYPHILIS OF THE NERVOUS SYSTEM

Syphilis begins to attack the nervous system in two ways: by round-cell infiltration and exudation affecting the blood-vessels of the meninges of the cord and brain; and by the development of a parenchymatous degeneration of the neurons.

Hence we have:

1. Meningo-vascular or exudative syphilis and
2. Parenchymatous or degenerative syphilis.

1. Meningo-vascular syphilis takes various clinical forms:
   (*a*) Cerebral syphilis.
   (*b*) Spinal syphilis.
   (*c*) Cerebrospinal syphilis.
2. Parenchymatous syphilis takes the forms of:
   (*a*) Paresis.
   (*b*) Tabes dorsalis.
   (*c*) Muscular atrophy.
   (*d*) Combined system degeneration.
   (*e*) Various combinations of the above.

Epilepsy and optic atrophy are added by Head and Fearnsides.

In many cases there is first the exudative and proliferative syphilis, attacking the blood-vessels and meninges, but in about half the cases this approach is so slight that the first noticeable effect of syphilis of the nervous system is a degenerative process. Syphilis begins openly to attack the nervous system in from two to twelve years after infection, but it may occur within six months or as late as thirty years. If it occurs early the meningo-vascular form dominates and may be the only one. If it occurs late, the parenchymatous and degenerative process is the chief and often the only one.

The causes leading the syphilis organism to attack the nervous system are a hereditary weakness or predisposition; abuse of the nervous system, especially in sexual excesses and in laborious occupation and, finally, the abuse of alcohol and food and the adoption of a mode of life which in general tends to weaken and exhaust the system.

There is some evidence that mild and hardly noticeable forms of Syphilis are those most likely to attack the nervous centres later. It is a fact that in about half the cases the patients deny either knowledge of any infection or of the development of secondary cutaneous symptoms (rashes, sore throat, alopecia, pains, etc.). This apparent immunity of the cases

with flagrant secondary symptoms may be due in part to the fact that these latter are more vigorously treated. The absence of any known primary lesion is explained by the existence of an infection without local reaction, or by its being masked by a gonorrhœa.

Of all persons affected by syphilis it is probable that in 2 to 10 per cent. the nervous system becomes involved. Men are rather more often affected than women with nervous syphilis, and rather more in proportion to the infection because they are more subject to the exciting causes. The conditions of modern civilization have increased the amount of nervous syphilis, and this increase is mostly in the degenerative types of paresis and tabes.

Symptoms.—Since syphilitic changes may attack any part of the nervous centres, the symptomatology of nerve syphilis is necessarily a varied one. The reader can perhaps best understand the way in which the disease acts by having presented, first, a series of tables showing on the one side the clinical symptoms, on the other the chief anatomical changes that underlie them.

Clinical Symptoms	Anatomical Change
I. PRODROMAL SYPHILIS.	
II. MENINGO-VASCULAR SYPHILIS (Hereditary and Acquired).	
(*a*) Of the Brain.	
Severe headache, vomiting, vertigo, mental dullness, and irritability, attacks of somnolence or coma, convulsions, cranial-nerve palsies, optic neuritis, hemiplegia, brain stem and bulbar palsies.	Syphilitic meningitis, arteritis and phlebitis.
(*b*) Of Brain and Cord.	
Many of the brain symptoms as above, spastic paraplegia, etc.	Meningitis; diffuse, disseminated, or localized meningo-myelitis.
(*c*) Of Spinal Cord.	
Paraplegia, Brown-Séquard paralysis. Muscular atrophy. Spastic paraplegia and ataxia.	Meningo-myelitis, gumma, localized softenings from obliterative arteritis.
(*d*) Of the Nerve-roots and Nerves.	
Cranial-nerve palsies, cauda-equina symptoms, local palsies of peripheral nerves. Muscular atrophy.	Root neuritis, gummatous neuritis.
III. PARENCHYMATOUS SYPHILIS (Hereditary and Acquired).	
(*a*) Of the Brain.	
Paresis.	Meningo-encephalitis.
Bulbar palsy.	
(*b*) Of the Cord.	
Tabes dorsalis.	
Spastic tabes.	
Muscular atrophy.	Meningo-myelitis.
(*c*) Of Brain and Cord.	
Tabo-paresis.	
IV. COMBINATIONS OF II AND III.	

I. **Prodromal Syphilis.**—The development of syphilis of the nervous system is often preceded by prodromal symptoms of a definite and recognized character. Such symptoms are especially apt to precede parenchymatous syphilis. They can be arranged somewhat as follows:

Meningeal Prodromata.—These consist of annoying and often severe headaches, temporary attacks of cranial nerve palsy; attacks of dizziness; dysæsthesiæ which are particularly referred to the head and back of the neck. They are symptoms which can be explained by the supposition that there is a slight grade of meningitis in some part of the brain or cord or else a slight degree of obliterative arteritis leading to local or general disturbances of the brain function.

Luetic Neurasthenia.—A most common group of symptoms is one which may be called luetic neurasthenia. The conditions is characterized by some mental depression, inability to use the brain effectively or long, annoying disturbances in the shape of paræsthesia of the head and neck and extremities, weakness of the sexual function, and the general group of symptoms which come under the description of neurasthenia of a rather depressive and often hypochondriacal type.

Luetic Deterioration.—In the milder forms of luetic neurasthenia, there is not so much an absolutely nervous exhaustion as a slight change in the character and in the effectiveness of the work of the individual. For a number of years before the development of a frank nervous lues, for example, the patient will have been recognized as having become a somewhat different person. He is not as attentive to his work or as keen in memory, or as efficient in his activities generally. He is more irritable and difficult to work with or to live with.

Luetic Melancholia.—Parenchymatous syphilis, particularly of the paretic or tabo-paretic type is not infrequently preceded by a definite attack of melancholia and this may be of very severe hypochondriacal or even suicidal character, running its course like other forms of melancholia. Much less frequently the infection is introduced by an attack of mania which may be mistaken for the onset of true paresis.

II. **Meningo-vascular Syphilis.**—*Of the Brain.*—In its most common form this shows itself by a gradual development of severe and persistent headache. This is usually associated with vertigo, sometimes with nausea and vomiting. After the headache has developed and has lasted for a time, or even without much delay, there comes on sometimes an attack of hemiplegia. Preceding the hemiplegia, or in some cases without the hemiplegia, there are paralyses of the cranial nerves, more especially of the nerves of the eye. Optic neuritis is somewhat frequent. The pupils may be irregular in outline and sluggish to light. There may be, before any paralyses develop, attacks of epileptic convulsions, either general or partial. Without any paralyses or with simply cranial-nerve paralyses, there may

develop attacks of somnolence and coma. Even if such attacks do not appear, the patient often shows a mental irritability and weakness, a slowness of the reasoning process, and incapacity to fix the attention such as is observed in connection with brain tumors, only with nerve syphilis these symptoms are not usually so marked. Polyuria and polydipsia are symptoms which are occasionally met with. The disease may take the form of a meningo-encephalitis producing symptoms of mental and physical disturbance not distinguishable from early paresis (meningo-vascular paresis).

Of Brain and Cord.—Very often some of the above groups of symptoms are associated with spinal disease. There is evidence of meningomyelitis, or of obliterating arteritis and as a result, we have paraplegia; or the meningeal syphilis of the brain may be associated with degenerative syphilis and we have lightning pains, ataxia and other signs of an occurring tabes.

Of the Cord.—The symptoms in spinal syphilis are commonly those of a transverse myelitis involving most often the lower part of the dorsal and upper part of the lumbar cord. This myelitis usually comes on rather slowly with the ordinary symptoms of a chronic or subacute transverse myelitis, there being a progressive paraplegia with spasticity of the legs and a good deal of pain. The condition is known as syphilitic spinal paralysis (see p. 230). Spinal syphilis may show itself also by the development of gummatous nodules which grow from the meninges, press upon the cord, and produce the symptoms of a spinal tumor. Spinal syphilis more rarely develops itself in three or four different foci, producing the symptomatology of disseminated myelitis. Finally, a spinal endarteritis may lead to focal hemorrhage and softening.

Pathology.—I have already given some indications of the pathological changes produced by syphilis. The disease affects the nervous system (1) by producing a meningitis with exudation, (2) by producing gummatous masses, (3) by producing an inflammation of the arteries, and (4) by so influencing the neurons so as to lead to their progressive degeneration. Of all these forms of anatomical change it is the arteries that are most often affected, and particularly the arteries at the base of the brain.

Syphilitic meningitis is characterized by the proliferation of round cells and the preponderance of an exudate which has a tendency to infiltrate into the nervous tissues. The anatomical characteristics of the syphilitic gumma must be studied in special text-books. The inflammation of the arteries attacks first the external coat and adventitia, producing there an enormous multiplication of round cells. The external coat becomes weakened, and as a result there develops beneath it, between the intima and the elastic layer, another exudate which constitutes what

is known as endarteritis. In syphilitic arteritis, therefore, there is both a peri-arteritis and an endarteritis; the former being usually the primary and most essential process. The endarteritis, however, as it develops gradually produces an occlusion of the arteries. This cuts off the circulation of the blood and leads to softening of the part. There is also a development of a hyaline degeneration in the arteries, which some regard as a very essential part of the anatomical change (Fig. 116).

Diagnosis.—The diagnosis of meningo-vascular syphilis is based upon the history of an infection, the irregularity and fugacity of the symptoms, a study of their peculiar clinical characters as described above. But all these things have been made of minor importance compared with those furnished by the reactions of the blood and the character of the cerebro-spinal fluid (see Serology of Syphilis).

The headache of syphilis is rather characteristic. It may attack any part of the head, but is usually unilateral or irregular, or again it may be bilateral in its distribution. The pain is very intense and sometimes exhibits a certain periodicity. It is not necessarily worse at night. It is apt to last continuously for from five days to three or four weeks. Headache of this character, followed by the paralysis of one or more cranial nerves or by an attack of hemiplegia, is extremely suggestive of syphilis. Optic neuritis is very liable to occur when the disease shows other evidences of being situated at the base of the brain. This optic neuritis is associated with contraction of the visual field, and a characteristic feature of this contraction is that it varies a great deal from week to week.

Prognosis.—Unquestionably the outlook is much more favorable than it is for any other organic disease of the nervous system. When the syphilitic process has not produced so much arterial disease as to lead to obliteration of vessels and softening, a very great degree of improvement and even a recovery may be expected. Headaches, cranial and other nerve palsies, paræsthesias and the neurasthenic and psychotic prodromata of exudative syphilis are usually curable. Lesions of the convexity are usually amenable to treatment. Syphilitic hemiplegia has a not much better prognosis than hemiplegia from other causes. Syphilitic myelitis has a not very good prognosis, but it is better than that of myelitis due to trauma. In all cases the results are enormously better the earlier treatment is instituted. Even bulbar and spinal paralyses, and meningeal or exudative paresis are curable or enormously relieved.

Treatment.—A laborious life full of worry and anxiety, in which the patient attempts to help himself along with stimulants, is surely provocative of nervous syphilis. These elements of cause must be removed.

In recent years the treatment of syphilis by hypodermic injections

of the bichloride or salicylate of mercury has been widely adopted, and better results have been obtained than by inunction or by administration by the mouth. One or two grains of the salicylate mixed with a liquid petroleum oil may be given twice a week. The effective treatment of nervous syphilis lies at present in the wise and persistent use of salvarsan combined as seems needed with mercury and iodides, tonics and hygienic measures. The details are given under Paresis.

Other drugs which are of value are the ordinary tonics, such as iron, quinine, and the bitters and mineral acids. Plenty of good food, outdoor air, and all those things which will improve the general health of the patient are indicated.

Syphilis of the Nerves.—Syphilis rarely affects the peripheral nerves; there are, however, occasional deposits of syphilitic exudate producing the ordinary symptoms of irritation and compression of nerves. There is said to be a form of multiple neuritis produced by syphilis, but its actual existence has not been demonstrated. Syphilis is sometimes known to attack the roots of the cranial nerves, producing a root neuritis. It attacks the roots of the spinal nerves when the spinal membranes are involved. In rare cases, where it involves only the anterior roots it causes a form of muscular atrophy.

III. **Parenchymatous Syphilis.**—The syphilitic parenchymatous processes lead to locomotor ataxia, general paresis, tabo-paresis, progressive spinal paraplegia, and sometimes to a form of muscular atrophy due to parenchymatous or vascular syphilis of the anterior horns.

Meningeal syphilis may be combined with parenchymatous forms, so that we sometimes see locomotor ataxia associated with meningo-myelitis; and we see paresis associated with meningeal syphilis.

Hereditary Syphilis.—Inherited syphilis will lead to anatomical changes and clinical manifestations resembling in all respects those of acquired syphilis. Inherited syphilis, in other words, may produce headaches, cranial-nerve palsies, hemiplegia, epilpsy, mental disorders juvenile paresis, feeble-mindedness, and degenerative spinal diseases. The disease probably is the cause of a considerable proportion of the cases of chronic hydrocephalus and of many of the cases of meningitis. The peculiarities of hereditary syphilis show themselves rather more in diffuse symptoms such as would be attributed to a meningitis of the convexity or circulatory defects; in other words, convulsions and mental weakness are rather more frequent, while hemiplegia and cranial-nerve palsies are comparatively rare. Hereditary syphilis also rarely attacks the spinal cord, although it is not unlikely that it is a factor in the production of some of the hereditary diseases of that organ. Hereditary syphilis develops at any time from birth to the eighteenth year, but most commonly under the age of five years. Serological tests furnish the diagnosis.

The presence of the Hutchinson teeth, the hazy cornea, and deafness or other ear trouble were the old diagnostic criteria of hereditary syphilis.

LOCOMOTOR ATAXIA

(Posterior Spinal Sclerosis, Tabes Dorsalis)

Definition.—Locomotor ataxia is a chronic progressive luetic disease, involving primarily the posterior spinal ganglia, roots and neurons, and later the spinal cord and peripheral nerves. It is characterized clinically by inco-ordination, pains, anæsthesia, and various visceral, trophic and other symptoms, and anatomically by a degenerative sclerosis chiefly marked in the posterior columns of the cord and posterior roots, and to a less extent in the peripheral nerves.

Forms.—Besides the common and typical form, there are anomalous and complicated types.

Types.
1. Common form.
2. Neuralgic.
3. Paralytic.
4. With initial optic atrophy.

Complicated forms.
With muscular atrophy.
With other scleroses.
With general paralysis.

Etiology.—The disease occurs oftenest in middle life, between thirty and forty, next between forty and fifty. It may occur as early as the tenth and as late as the sixtieth year. In the very early cases it is usually due to hereditary syphilis. It is much more common in males (about 10 to 1). Hereditary influence is only indirect; *i.e.*, the parents may transmit lues or a neurotic constitution. Diathetic influence is slight. Exposures to wet and cold, combined with muscular exertions, are effective causes. Soldiers, travelers and drivers are rather more susceptible. Excessive railroad traveling, excessive dancing with exposure, favor the development of the disease. Excessive sexual intercourse, combined with irregular living, is a predisposing cause.

Syphilis is the one important and essential cause. A personal history of the disease is obtained in from 60 per cent. to 90 per cent. of the cases. Serological tests show almost invariably evidence of syphilis and statistics indicate that about 1 per cent. of those infected with syphilis become tabetic unless treated actively and by modern methods.[1] Lack of

[1] The following statistics from my own experience show the physiognomy of the disease in a cosmopolitan American city. Total cases, 248; males, 226; females, 22. Ages when disease began: Average age at time of onset, 40; beginning a year or two earlier in private patients and in those with a history of syphilis and active anti-syphilitic treatment (Collins). Average period between infection and tabes, 10 to 20, ranging from one and one-half to twenty-five years. This is much longer than Erb's estimate, but is reached by three independent studies of my cases. Average duration

proper treatment for syphilis is believed to favor the development of the disease, but a careful study of the statistics of my clinic and of his own cases by Dr. Joseph Collins seems to show that antisyphilitic treatment *as formerly carried out* does not prevent the disease if the other favoring conditions, such as exhausting work and sexual or alcoholic excesses, are present.

Among exciting causes are profoundly depressing emotions, acute infective diseases like typhus, pneumonia and rheumatism, difficult labors with severe hemorrhage, prolonged lactation and excessive smoking. Edinger lays great stress on the over-use of the limbs. Trauma as an apparently exciting cause is noted in a very small percentage of cases (less than 1 per cent.). In my experience there has always been evidence that the disease was already in the system but had not been recognized in these cases.

Symptoms.—The disease is generally divided into three stages: the initial or pre-ataxic, the ataxic and the paralytic.

1. The pre-ataxic or initial stage. Previous to any other notable symptoms, the patient may have for months or years attacks of sharp pains darting through his limbs or about his trunk. With this he may have only Argyll-Robertson pupils and loss of knee-jerks and ankle-jerks or of ankle-jerks alone. To this may be added a little uncertainty of gait. Or instead of the pains he may have for a long time gastric crises. Occasionally nothing is noted at first but loss of sexual power or a weak bladder.

A gradual development of optic atrophy and blindness may also precede nearly all other symptoms. Ocular palsies and hypotonia may also be among the preataxic symptoms. In many cases the disease does not advance beyond the preataxic stage, and this is particularly true of neuralgic and optic forms of tabes.

When the ataxic process sets in, in addition to the symptoms mentioned the patient notices a slight uncertainty in walking, especially at night; he has numb feelings in his feet. His sexual function becomes weak, his control over the bladder slightly impaired. He has temporary attacks of vertigo and of double vision. A sense of weariness oppresses him, even though he has made no exertion. He may have attacks of vomiting and gastralgia, and may suffer from constipation, with hemorrhoids and rectal pains. Arthropathies may appear. Some hypotonia may be present. Such symptoms may last a few months or several years; often the disease is kept in this stage.

2. The ataxic stage. The gait now becomes so unsteady that others

of disease when seen by me, eight years; average duration of life in five fatal cases, twelve years, ranging from five to twenty years. Complications: optic atrophy, 6 per cent.; marked arthropathies, 5 per cent.; with general paresis, 4 per cent.; paraplegia, 1.5 per cent.; hemiplegia, 1.6 per cent.; eye palsies, 8 per cent. About 10 per cent. of 300 private cases were complicated with paresis—tabo-paresis. The complication is smaller in dispensary cases. High tabes (arm tabes), 5 per cent.; cranial nerve palsies, 10 per cent.; ophthalmoplegia in 1 case, progressive muscular atrophy in 3, laryngeal palsies in 3.

notice it; the patient has to have help, and when walking he has to watch his feet and the ground. If he stands with his eyes closed, he totters and may fall. His feet feel as though there was a layer of cloth or cotton between the soles and the ground. Paroxysms of lightning-like pains continue to attack the legs, chest and arms; tests show anæsthesia of the toes and feet or in areas on the legs and on the trunk. A sense of constriction is felt around the waist. The sexual power is lost; the bladder is weak, and care has to be taken to empty it. The bowels are constipated; at times he has attacks of intense pain in the epigastrium, with vomiting and perhaps a diarrhœa coming on without cause. The pupils

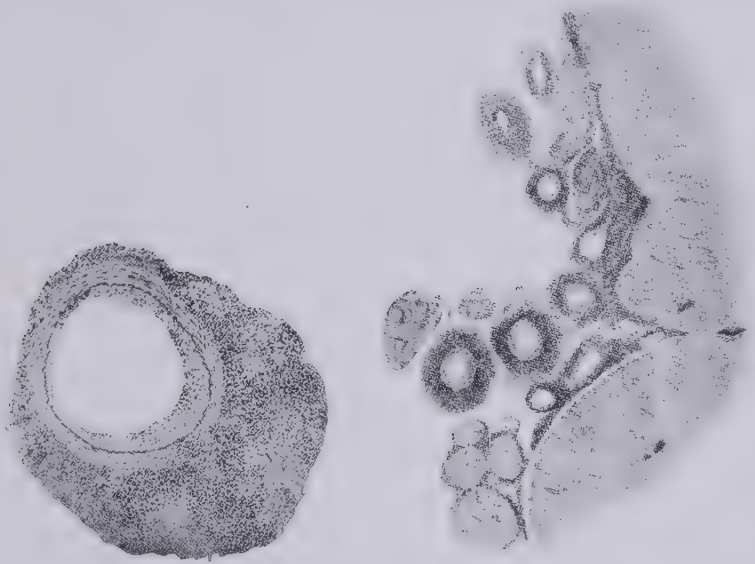


Fig. 115.—Meningo-vascular disease in syphilis of brain and cord. (*Bruce.*)

are small often unequal and irregular and do not react to light, but do react to accommodation; vision is still good. The incoordination and pain and anæsthesia after a time affect slightly the arms. The muscles become relaxed and atonic. The face shows atonia; also there is often a pseudo-ptosis of one or both lids. This stage lasts several years.

3. The paralytic stage. After several years with various remissions and improvements, the patient loses altogether the power of walking. His legs are somewhat wasted, but the muscular strength is fairly good. The anæsthesia and ataxia are very great. The patient does not feel the prick of a pin or touch of the hand; nor with closed eyes does he know where his legs are. His bladder is anæsthetic and paretic, so that the urine has to be drawn. The pains are much less, but are still present at times. The arms are more involved, but never so seriously as to make

them useless like the legs. The intelligence remains good, and the patient may continue bedridden for years, dying finally from some intercurrent affection.

The following table shows the prominent symptoms in the usual order of their appearance:[1]

	First stage (half to thirty years)	Second stage (two to ten years)	Third stage (two to ten years)
Motor	Eye palsies.	Less.	
	Ataxia.	Increased.	Increased.
	Muscular weakness and hypotonia.	Paresis, increased.	Paraplegia.
		Increased.	Increased.
Sensory	Pains.	Pains.	Painless.
	.....	Anæsthesia.	Increased.
Excito-reflex	Loss of knee-jerk.		
	A.-R. pupil.		
Trophic	Arthropathies.	More rare.	Rare.
Visceral	Sexual weakness.	Increased.	Increased.
	Vesical weakness.		
	Constipation.		
	Gastric crises.	Decreased.	Decreased.
Special senses	Diplopia.	Rare.	
	Optic atrophy.	Rare.	Increased.
	.....	Deafness.	Increased.

Analysis of Symptoms.—A study of the symptoms in tabes produces enormous results in the way of clinical phenomena. The student should not, however, lose his sense of proportion in going over these data. After all, the serious things in tabes are not so numerous. They are his ataxia and loss of ability to walk well, his pains, sexual and bladder weakness, hypotonia, anæsthesia, crises, especially gastric, arthropathies, and optic atrophy.

Locomotor and *static ataxia* are present very early, but only to a moderate extent. Tests, such as making the patient walk and stand with the eyes closed, noting the position of limbs and the weight of objects, will reveal an ataxia due largely to beginning anæsthesia of the joints and tendons.

[1] The disease attacks different levels of the cord in about the following proportion: Optic tabes, 5 per cent.; high (arm) tabes, 5 per cent.; dorsal (abdominal and trunk) tabes, 20 per cent.; lumbar and sacral tabes, 60 per cent. To these add tabo-paresis, 10 per cent.

The patella-tendon reflex or knee-jerk is abolished very early in all typical cases. This constitutes a very important symptom, therefore. The *ankle-tendon* reflex is lost early in the disease, but not, as a rule, so early as the patella reflex. Inequality or diminution in the activity of these reflexes are significant when taken in connection with other symptoms. An inequality is more important than a general diminution. These reflexes of knee and ankle may disappear early in the disease and return under treatment, but this is rare and later they may disappear again.

Fig. 116.—Showing station in second stage of locomotor ataxia.

The gait and station in ataxia are characteristic. In walking, the patient keeps his eyes on the ground and on his feet. The latter he throws out rather forcibly, owing to over-action of the extensors of the foot. In watching such a patient walk barefooted, the extensor tendons can be seen to stand out with each forward movement of the limb. The foot is brought down sharply on the heel and the legs are spread apart a little. Turning a corner, turning around and going downstairs are done awkwardly, and the patient is apt to totter and fall. Walking on a chalked line is very difficult; so also is walking backward. The gait improves after the patient walks a while, and he will generally say that the practice of walking does him good. Still, he soon gets tired (Fig. 116).

Hypotonia, or relaxation and lengthening of the tendons and muscles, is usually an early and always an important symptom, but is sometimes not very marked until later in the disease. This is what causes the *genu incurvatum;* when present in high degree it is an unfavorable sign, for it is apt to lead to deformities and promote arthropathies (Figs. 118, 122).

Myoclonic and tic-like spasms may affect the face and body.

Myokymia (fibrillary and wave-like contractions of the muscles) is

an early and rare complication. It occurs usually in cases with rather rapid onset, and affects most the muscles of the lower limbs. Though not painful, it is a very annoying phenomenon.

Severe rectal *neuralgia*, associated perhaps with hemorrhoids, is sometimes an early symptom. Persistent neuralgia and functional disturbance of the bladder and rectum should cause suspicion of ataxia. Lancinating or lightning pains occur and are very characteristic. The pains dart down the legs along the course of the sciatic, or they suddenly appear as patches of pain on the foot or leg or thigh ("spot pains"). The pain comes unexpectedly and with such severity that the patient involuntarily jumps or jerks the limb. He speaks of his "jerking" and "twitching pains." The pains may affect the bowels or be felt as a squeezing sensation around the waist (girdle pains). The pains of ataxia are often the most obstinate and distressing symptom. They usually come on in great intensity once or twice a month, and last for two or three days. They then leave the patient for a time. They are often worse in cold and damp weather. In some cases the pains are almost continuous, coming on, if not every day, at least two or three times a week. Such cases are associated with much cutaneous hyperæsthesia, especially during the attacks. This type of cases is called "*the neuralgic.*" The patients rarely have as much ataxia, paresis or visceral troubles as the typical forms present, and in certain respects such cases are favorable. Sometimes the pains are accompanied with erythema or herpes zoster showing an acute involvement of a posterior ganglion.

Fig. 117.—Hypotonia in late tabes dorsalis.

The pains of the disease continue well into the second and even third stage. Meanwhile the anæsthesia becomes much more marked. It affects most the feet and next the legs, rarely extending much over the thighs, but passing to the fingers and hands. The anæsthesia is greatest to pain, but touch and temperature sense are also involved. There is often delayed conduction and polyæsthesia; and many other curious perversions of the cutaneous sense are noted. Some anæsthesia usually develops over the finger tips and hands, and sometimes a band of anæsthesia develops about the trunk (Fig. 119). This trunk anæsthesia may be one of the early symptoms of the disease. The facial and cranial nerves are not much affected, but there may be trigeminal neuralgia. The olfactory

nerve is occasionally affected. In early stages of the disease when there is perhaps some specific disorder of the mucous membrane, parosmia occurs, the patient being annoyed with the sense of a bad smell. Later in tabes this sense may be lost.

Optic atrophy occurs in about 6 per cent. of cases in my experience. Optic atrophy usually develops in the preataxic stage, and if a patient has reached the *second stage without it, he will probably escape it altogether.* Cases with ocular paralyses are slightly more disposed to it (Berger). It attacks the left eye oftener than the right. The atrophy begins sometimes with increased sensibility to light, flashes of light, and muscæ volitantes.

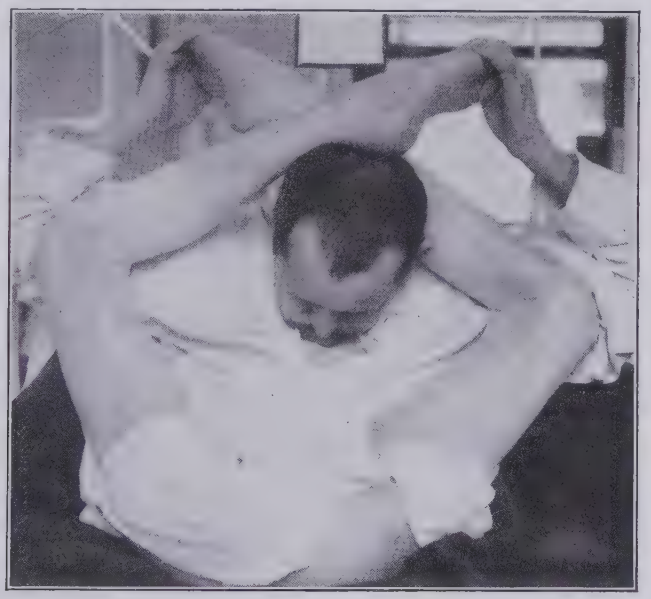


FIG. 118.—Hypotonia in locomotor ataxia.

With the failing vision, disturbance of color sense often and contraction of the visual field always occur. This contraction is irregular, with sector-formed defects; not hemiopic. The atrophy progresses slowly with slight remissions. It may cease its progress, but this is rare and blindness comes in about three years. Ophthalmoscopically, there may be seen slight evidence of congestion in the early stage; later, pallor of the discs, which finally become grayish.

The *eye muscles* are implicated in some way in nearly all cases of tabes. The following are the disorders: (1) Loss of the light reflex and myosis; (2) sympathetic-nerve ptosis; (3) paralysis of the external eye muscles.

Paralyses of the ocular muscles (third, fourth and sixth) occur rather oftener in cases with evidence of exudative (meningeal) syphilis. Ocular palsies are early symptoms of the disease, occurring, as a rule, in the preataxic stage.

1. Loss of light reflex and pupillary rigidity. The pupils are small and sometimes uneven and irregular in shape; they respond to accommodation, but not to light. In early stages the light reflex may be simply sluggish. In the late stages the pupils are often dilated. The

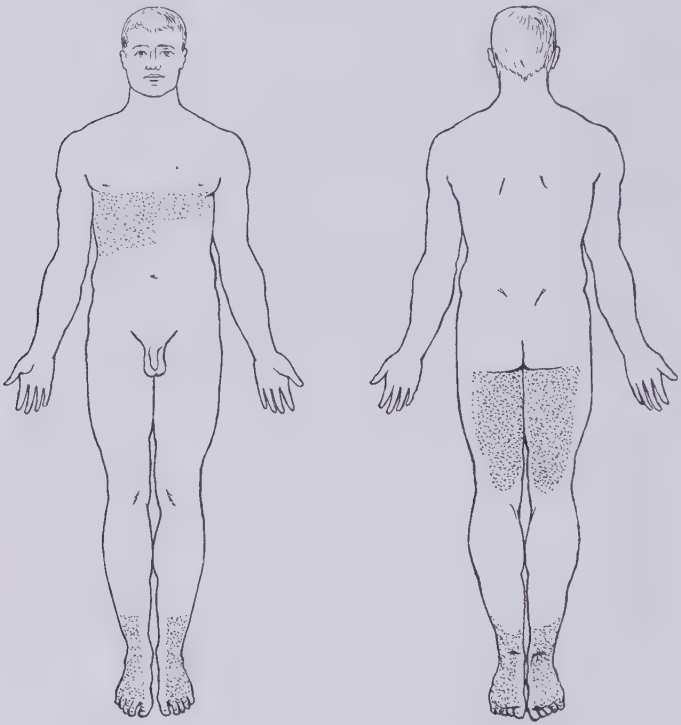


Fig. 119.—Trunk and leg anæsthesia in early stage of tabes dorsalis.

phenomenon is perhaps due to lesion of the ciliary ganglia. More probably it is due to degeneration of the mesial nucleus of the third nerve, or its connections. The Argyll-Robertson pupil is practically found only in tabes and in general paresis. It may be brought about by local disease, by a polioencephalitic or ganglionic infection, or it may be congenital. The ocular skin reflex usually disappears early. The myosis in tabes is due to paralysis of the sympathetic dilating fibres.

2. Sympathetic nerve ptosis. A slight drooping of one or both lids is not infrequent. It begins early and progresses slightly up to the later stages of the disease. It is due to paralysis of the cervical sympathetic-

nerve fibres which go to the unstriated fibres of the levator palpebræ (tarsalis superior).

3. Paralysis of the external eye muscles. The external rectus is oftenest affected of single muscles, but the various branches of the third nerve taken together are oftener involved than the sixth. Of the third nerve's branches, the levator palpebræ and internal recti muscles are oftenest involved. There may be multiple palsies. These occur oftener with meningeal syphilis. Progressive ophthalmoplegia may be associated with tabes. The ocular nerve palsies may be transitory or permanent. Those occurring in the preataxic stage are usually transitory, lasting a few hours, days or weeks. Cases have even lasted two years and got well. The permanent palsies develop usually in the later stages. The early palsies are usually due to a syphilitic exudation at the base of the brain; the late palsies are usually due to degenerative lesions of the nuclei of the ocular nerves.

Violent neuralgic pains sometimes occur in the face, but it is rare; in fact, a characteristic of tabetic pains is that they hardly ever involve the *trigeminus*. However, it does occur that a typical tic douloureux may develop.

Paralysis of the *seventh nerve*, though not infrequent in cerebral syphilis, is not a symptom in tabes. It is not even a premonitory symptom as are the ocular palsies. Spasmodic movements of the lips and face with smacking movements similar to those seen in some forms of spasmodic tic have been noted in two of my patients.

The *acoustic nerve* is frequently affected, some form or degree of deafness occurring in about one-fourth of the cases; but in the majority of instances the trouble is an accidental complication due to middle-ear disease. Primary atrophy of the auditory nerve is very rare, as might be expected, since this nerve is structurally not like the optic nerve. Its existence has been inferred on clinical grounds. There is a form of tabetic deafness of trophic origin due to a sclerotic condition of the middle ear (Treitel). The labyrinthine nerve is affected but rarely. Vertigo is not common, yet labyrinthine crises causing seizures like those of Ménierè's disease have been observed.

Dysæsthesia of the throat with attacks of repeated swallowing movements form what Oppenheim calls pharyngeal crises.

Bulbar paralysis as well as progressive muscular atrophy are rare association diseases and generally appear late in the course.

More common are disorders of innervation of the larynx. These consist of *laryngeal crises* and paralyses of the laryngeal muscles. The characteristic paralysis of tabes is a "posticus palsy;" *i.e.*, a paralysis of the posterior crico-thyroid muscles. These are abductors, and their paralysis causes difficulty in breathing if both are affected and sometimes

when one is affected. The paralysis is usually unilateral. The laryngeal palsies are usually early symptoms and usually are not serious. I have known, however, sudden death to occur in a patient subject to attacks of this type of palsy. Anæsthesia of the throat and larynx is very rare.

In a laryngeal crisis there is spasm of the adductors or paralysis of abductors, with noisy, croupy respiration. The attacks come on suddenly, the patient coughs and struggles for breath, and he may be seized with vertigo and fall down. The pulse may be very fast. The paroxysm lasts for a few minutes to several hours. The symptoms are very distressing, but not usually dangerous.

Paroxysms of cough have been described as "bronchial crises."

There are also *cardiac crises*, in which there are dyspnœa and rapid heart beat and sense of suffocation resembling angina. A much more frequent symptom is a rapid pulse which runs about one hundred and is small and weak.

The heart is sometimes diseased, either from neurotrophic disturbance or luetic degeneration. The pulse is often small, rather rapid, and weak.

The most common and distressing of the crises of tabes are the *gastric*. These sometimes occur in the preataxic stage and recur for a long time before the nature of the trouble is recognized. They consist of sudden attacks of intense pain extending from the groin to the epigastrium or encircling the waist, accompanied by vomiting and sometimes diarrhœa. The attacks are often associated with pains in the legs. They last two or three days, then usually pass away, but some cases last two or three weeks and leave the patient much exhausted and often more ataxic. Sometimes instead of or between the crises the patient has a continual diffuse, dull, abdominal pain.

The *arthropathies of locomotor ataxia*. Degenerative diseases of the joints, technically known as arthropathies, and spontaneous fractures of bones form important symptoms of tabes. They occur in 10 per cent. (Charcot) or 5 per cent. (author) of cases.

The arthropathies are three or four times more frequent than the fractures. The joints oftenest affected are the knees, ankles and hips; but the elbow, shoulder, wrist, vertebral and pelvic bones and small joints may be attacked.

Spontaneous fractures occur oftenest in the shaft and neck of the femur, next in the legs, forearm, humerus, and clavicle. The pelvis, scapula, vertebræ and under jaw may be fractured. Arthropathies are often accompanied by fractures, especially of the heads of the bones. The two sides of the body are about equally affected.

The arthropathies are characterized by a sudden, apparently spontaneous painless swelling of the joint. The symptoms may develop in

twenty-four or forty-eight hours. In rare cases there is a history of some preceding rheumatic pains or of an injury. After a time there is an osseous hyperplasia of the joint, which becomes enlarged to enormous proportions. There is also a tendency to luxation of the joint. It crepitates on moving. There is no tenderness on pressure; the hand finds evidence of synovial exudation, roughened surfaces, and perhaps fractures of the enlarged parts. In the milder forms there are simply swelling from synovial exudation and some enlargement of the bones with roughened surfaces. After a few weeks this swelling may subside and the joint return to nearly its natural size. In other cases the process progresses, the ligaments relax, the bones of the joint can be moved about freely, and luxations are easily produced. There is still no pain, but the limb becomes almost or entirely useless on account of the loose and relaxed condition of the parts (Fig. 121). As time goes on, some absorption takes place and the head of the bone may almost disappear. The arthropathies have been divided into benign and malignant, but no sharp line can be drawn or certain prognosis made in the early stage. The arthropathies appear in the prodromal and early stage of the disease in over half the cases, and are often at first unrecognized. One-third occur after the tenth year of the disease.

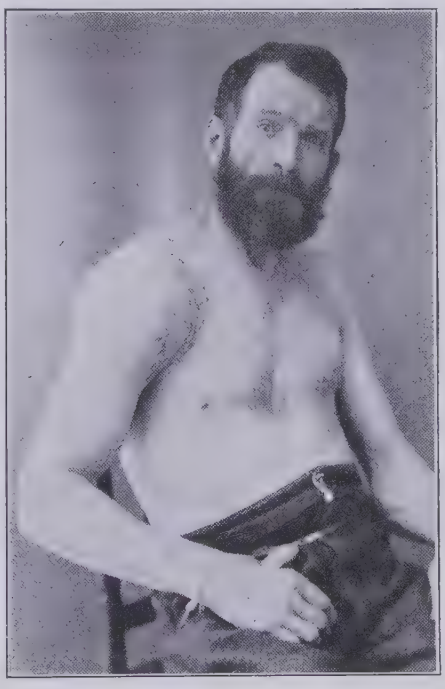


FIG. 120.—Arthropathy of shoulder.

The spontaneous fractures are usually brought on by a slight trauma, such as a fall. Violent muscular movements may produce them. They also are painless, as a rule. The fractures usually heal well, often with abnormal readiness, but occasionally there is delay, and often healing is accompanied by great throwing out of callus.

Pathologically, the arthropathy is a rarefying osteitis. It does not differ anatomically from arthritis deformans, except that fractures may accompany it. Clinically, the chief difference lies in the abruptness, spontaneity and painlessness of the process. The disease, on the whole, cannot be considered specifically different from arthritis deformans,

modified by the analgesia of the parts. It is due probably to a degenerative change in the nerves supplying the joints and bones. The process may begin in the cartilage, bone or ligaments. Eventually all these parts are involved. There is congestion of the synovial membranes with hydrarthrosis, then atrophy and rarefying hypertrophy of the epiphyses, relaxation of the ligaments, formation of osteophytes and bony stalac-

Fig. 121.—Arthropathy of knees.

tites. There may be a rarefying osteitis of the long bones, without much joint involvement at first.

Various *trophic disturbances* of the skin may appear, generally late in the disease. The most common are herpes and lichen. Besides these, bullæ, transitory erythema, urticaria, eczema, pemphigus, ecthyma, ulcers, ichthyosis and petechiæ have been described; but they are rare

and often only accidental complications. A peculiar round perforating ulcer sometimes develops on the sole of the foot, often as the result of cutting a corn. In rare cases the nails and teeth fall out. In frankly syphilitic cases there is usually baldness. The skin of the tabetic patient loses its "tone" due probably to the general muscular atony and to weakness of the erectores pilarum. At any rate, the hand has a characteristic flabby feeling which is almost diagnostic of the disease. The face and cutaneous tissue generally show the same characteristic.

A *sense of great weariness* and heaviness in the limbs, present constantly, no matter how much rest is taken, is a characteristic early symptom, and is due to an irritability of the nerves of muscular sensibility.

Muscular atrophies occur sometimes in tabes. They are of three kinds: (1) a progressive muscular atrophy due to local endarteritis and resulting degeneration of trophic and motor cells (chronic poliomyelitis); (2) localized muscular atrophies due to degenerative atrophy of nerves; (3) a general wasting. Under the first head one finds ophthalmoplegia, bulbar paralysis and spinal myopathies; under the second, wasting of certain groups of muscles in the legs or arms.

FIG. 122.—Perforating ulcer of foot.

Besides these, there is a generalized atrophy which occurs in the paralytic stage and is due probably to a slight involvement of the anterior horns in the progressive process that affects the cord.

Attacks of *hemiplegia* in rare instances occur in tabes. They are usually of temporary character and occur early in the disease. They may come on late and are then more likely due to acute softening due to disease of the cerebral vessels of syphilitic origin.

Acute paraplegia comes on occasionally also, and this sometimes almost disappears.

The *sexual power* may be at first greatly exaggerated; but this is rare, and usually there is progressive weakness and loss of desire. The bladder and sexual functions are rarely entirely lost and rarely equally impaired in the first stage; one may continue good while the other is affected moderately. Usually the sexual function goes first.

Some *cerebral symptoms* occur in tabes, chiefly in the early stage. They are insomnia, which may be very obstinate, and occasional vertigo. An irritability of temper and tendency to despondency, sometimes noted,

cannot be considered unnatural. Apoplectiform and epileptiform attacks are described, but are very rare, and should cause a suspicion of a complication. The disease in very rare cases terminates in general paresis. Usually if there is to be paresis it comes on with the tabes. When the disease developes frankly and typically as locomotor ataxia, paresis rarely supervenes.

Course.—The disease has been termed progressive, but it is not so in a large number of cases. With proper treatment the symptoms can often be kept in control for years. The first stage may last twenty years or more; the second stage five to fifteen years. The total duration of the disease varies enormously, ranging between three and thirty years. A few acute cases have been observed, running a course of less than a year.

Complications.—These are acute myelitis, generally syphilitic; lateral sclerosis, progressive muscular atrophy, hemiplegia from embolism or endarteritis, general paresis and heart disease.

Tabo-paresis.—In a rather increasing number of cases one sees the symptoms of paresis and tabes develop together. In these cases the cerebral symptoms are usually more dominant and are always more serious and important. These symptoms do not differ greatly from the ordinary early symptoms of paresis. The tabetic symptoms are often of less note and consist in loss of knee-jerks, some ataxia, lancinating pains, ocular palsies and vesical and sexual weakness. Taking a group of 100 cases of tabes I find about 10 per cent. are tabo-paretic. In these cases it is the paresis that has to be more carefully watched and dealt with. Like tabes, it can be arrested, but not so often as in the pure type.

Pathological Anatomy and Pathology.—The characteristic changes in an advanced case are found in the spinal cord, posterior spinal ganglia and posterior roots, and to a less extent in the peripheral nerves. The spinal cord usually is reduced in size and flattened antero-posteriorly; the pia mater is thickened somewhat. One can see with the naked eye that the posterior columns of the cord are shrunken and have a grayish appearance.

In early cases one sees that the part of the posterior column first affected is a vertical streak lying in the middle root zone between the posterior median (columns of Goll) and posterior external columns (columns of Burdach) (Fig. 123). The segments first and most affected are those of the upper lumbar and lower dorsal region. Besides this area the rim zone or column of Lissauer is also early involved. As the disease progresses it extends upward and spreads laterally so that finally all of the posterior column is changed into a dense connective-tissue mass through which only a few nerve-fibres run. The part last and least involved is that lying just posterior to the commissure (anterior root zone of Flechsig, ventral fundamental column) and that lying just

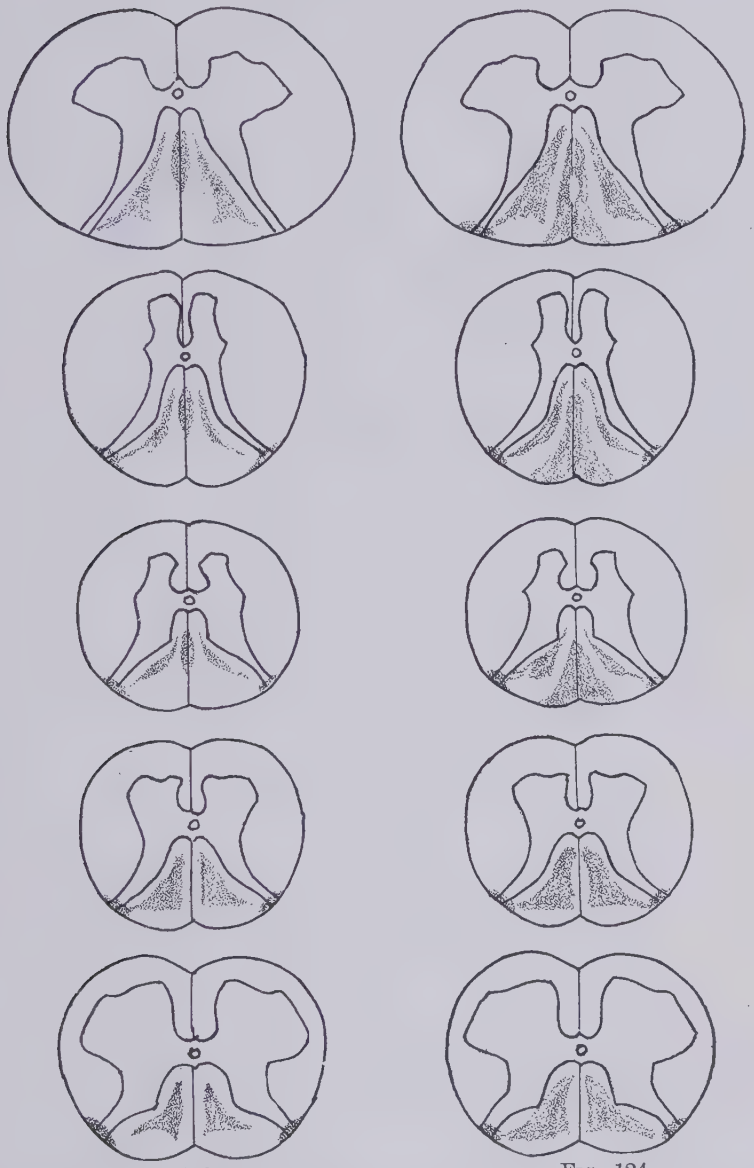


FIG. 123. FIG. 124.

FIG. 123.—Locomotor ataxia, showing areas affected in first stage at five different levels. Drawn from specimens in author's possession and from comparative study of over thirty other figures.

FIG. 124.—Locomotor ataxia, second stage.

mesial of the posterior horns (external part of the middle root zone) (Fig. 125). There is sometimes a degeneration of the antero-lateral ascending tract (Gowers' tract), and very rarely of the cerebellar tract. The pyramidal tracts are involved only in complicated cases. The cells and fibres of the column of Clark are often involved in advanced cases. The gray matter of both the posterior and anterior horns may show some degenerative changes, viz., decrease of the fibre network and atrophy of the cellular elements.

The posterior roots are usually involved, the process extending as far as the spinal ganglia, which also show some degeneration, but the lesion is not strikingly marked here in all cases, and sometimes the spinal ganglia are nearly healthy, although the posterior columns are diseased. The anterior roots are fairly normal.

The exact initial point of attack varies, and this accounts for the variation in the symptoms. Cases that begin with decided bladder and genital symptoms probably start low down; cases which go for a good while with only ataxia, loss of knee-jerk, and pains begin higher; while in the brachial or arm-type cases the process begins in the cervical enlargement.

The peripheral nerves are diseased in a large number of the advanced cases. The nerves of the leg are most involved. The process is a degenerative atrophy or neuritis. It affects the extremities of the nerves first and slowly extends upward, seldom reaching the large trunks.

Sometimes the disease begins in the optic nerves, but not in the bipolar visual cells of the retina which correspond to the posterior ganglion cells. The process here is an atrophy beginning at the periphery and extending brainward. The third, fifth and sixth nerves are occasionally involved; still more rarely the olfactory and auditory. The vagus nerve and sometimes its nucleus and that of the glossopharyngeal are implicated, it may be, rather early in the disease. It is believed that these facts explain many of the laryngeal and visceral crises and the tachycardia.

Pathology.—Syphilis often invades the nerve-centres before true tabes sets in and frankly shows itself in the form of inflammatory, meningeal, vascular and gummatous exudates, but often it does not betray its presence in this way but does its fatal work quietly through years of apparent health. All the time its poison is at work instituting a tendency to death and degeneration in certain parts of the nerve-centres. The parts which are usually first selected are the posterior spinal roots and the neuraxons which pass from the nerve-cells of the ganglia into the posterior roots and columns of the spinal cord. Locomotor ataxia, therefore, is not primarily a disease of the posterior spinal columns, but of the peripheral sensory neurons. It is true that the posterior spinal ganglia are not always so seriously diseased as the posterior columns of

the cord. This, however, is because the severity of the disease is first shown in the central neuraxons of the ganglia and their collaterals, just as in alcoholic neuritis the peripheral parts of the motor neuraxons are most and earliest affected by alcohol. The *spirochæta* toxin is carried in the peri-radicular spaces and starts often a radiculitis or root

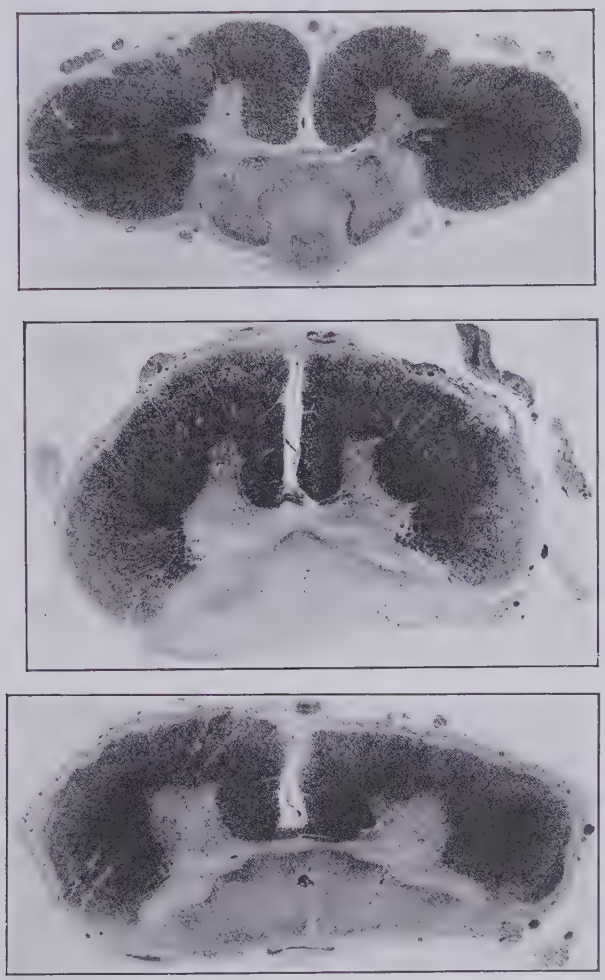


FIG. 125.—Tabes dorsalis, advanced stage; cervical dorsal and lumbar levels.

degeneration, which extends into the cord. As the disease extends, it involves both the peripheral and central parts of these sensory neurons; that is to say, both the sensory fibres and the posterior spinal roots. Still later it attacks other portions of the nerve-centres. The syphilitic microbe and its toxins are brought to the nerve-centres in the blood,

whence may pass into the lymphatic sheaths of the blood-vessels or are thrown out upon the serous membranes in the subdural sacs. In the circulation of the cerebrospinal fluid the toxins are carried along the serous sheaths which surround the cerebrospinal nerve-roots. The nerves, as they pass out from the spinal cord, are covered with three membranes; the dura mater, the arachnoid and the pia mater. The dura and the arachnoid surround them less tightly than the other. The dura becomes fused with the connective tissue supporting the nerves as they pass out, and the arachnoid becomes fused with the epineurium and perineurium. Now, fluid injected into either the subdural or subarachnoid spaces passes readily along the nerves for some distance (Macewen), and syphilitic toxins in the subarachnoid and subdural spaces of the spinal cord will thus have a tendency to infiltrate along these sheaths, but as it passes out of the vertebral canal or cranial cavity it meets mechanical obstacles, owing to the constriction of the parts, and there is therefore a certain damming up or accumulation of the poisonous material at these points of exit. Generally at this point it meets with the posterior spinal ganglion a highly organized structure with special vascular supply, and it therefore naturally deposits its poison upon this part, which furnishes much more opportunity for mischief than the non-vascular anterior roots. It is a fact that many of the initial symptoms of locomotor ataxia are thus connected with such troubles as would occur from an exudate trying to get out along the course of the spinal or cranial nerves. A frequent initial symptom, for example, is palsy of one of the third nerves or of the sixth or seventh nerves, due to exudates clinging around their roots. Still more frequently the initial symptom is a neuralgic pain in the course of the sciatic plexus, due to the effects of this poison upon the ganglia. It has been established that the syphilitic infection is due to the *spirochæta pallida*. These organisms have been found in tertiary forms of syphilis, and it has been shown that the *spirochæta* is alive and acting upon the nervous system, even in degenerative or parasyphilitic stage. Thus the degenerations of tabes are really only a later form of a constantly acting infection and all the phenomena, both of syphilis proper and so-called parasyphilis are only different stages of the same pathological process.

I assume in this description that it is a syphilitic poison which is always at work as a cause of tabes dorsalis. This, however, may not necessarily be the case, for it may be the infection of other diseases; and the results of other poisons, such as ergot, pellagra, etc., may be explained in the same way. There is an inadequacy of the lymphatic and venous systems thoroughly to rid the spinal canal of the poisons that lie in it; the body of the cord is cleared, but the roots do not get rid of the poison.

As to why this process attacks some persons and not others, it can only

be said that certain people are born with defective power of resistance as regards their nerve-centres, and that others induce this defective state by physical and other excesses.

Diagnosis.—In the first stage the disease has to be distinguished from hereditary ataxia, multiple neuritis, chronic myelitis, spinal tumor, spinal syphilis, general paresis and neurasthenia. In hereditary ataxia the age, the history of the disease, and the absence of lightning pains are usually sufficient to distinguish it. Multiple neuritis, in its sensory or pseudo-tabetic form, sometimes resembles closely locomotor ataxia. The differential points are given in the sections devoted to that disease. In myelitis there is more paralysis, generally exaggeration of reflexes, and an absence of disturbance of special senses.

The serological formula in tabes varies, but it is about as follows:

Ser. Wassermann	− or +
Cer. spinal fluid Wassermann	+ or −
Globulin	+
Cells	30 to 50

This formula is changed to negative by active and thorough treatment. It becomes negative in very advanced periods of the disease. (See Paresis.)

The diagnostic criteria of locomotor ataxia in all cases are the presence of lightning pains, numbness of the feet, loss of knee-jerk, hypotonia, arthropathies, ataxia of station and gait, without much loss of muscular power, the presence of the Argyll-Robertson pupil, the history of syphilis and the slow onset of the disease. These criteria apply especially for the early stages of the progressing disease. *A lost knee-jerk, lightning pains, a positive serological formula* and *stiff pupils* are usually quite enough to assure a diagnosis.

Prognosis.—In the first stage a large percentage may have the disease stopped and get practically well; and tabes dorsalis, if seen early and properly treated, is no longer a progressive disease. In the second stage a cure is impossible, but great improvement may be secured and the patient made relatively comfortable for years.

In the third stage little can be done except to relieve the symptoms, but life may be prolonged. In some cases the careful application of mechanical therapy (the Frenkel exercises) will enable patients to walk to some extent again. The malady is of course better controlled if the patient can afford to lead a regular and quiet life. This is one of the diseases in which the poor man has not as good a chance, because hospitals will not keep him long enough.

Death usually occurs from some intercurrent malady or from kidney

disease caused by the bladder trouble. Patients very rarely indeed die from the disease itself and its various "crises."

Treatment.—The treatment of locomotor ataxia is a subject the discussion of which cannot be made dogmatic, for the treatment depends very largely upon the patient and the stage and type of the disease. My experience is that any treatment depends enormously upon one's opportunities of getting the patient in the earliest stages. Supposing this be done, the first thing is to be quite assured that there is no trace of syphilis in the system as shown by clinical signs and by tests of the blood and cerebrospinal fluid.

It is the fashion now to begin treatment by giving intra-venous or intra-spinous injections of salvarsan. Brilliant results have been secured and perhaps better or more uniform results than from the use of mercury and iodide. Injections of neo-salvarsan 0.9 may be given twice weekly until ten doses have been given. In the intervening days inunctions or hypodermics of mercury should be given in average doses. After this, hypodermics of salicylate of mercury, gr. i. may be given twice a week for six weeks. Then the patient should be given tonics and general hygienic measures. If at the end of two months, the serological formula is not markedly improved another course of injections is given. If the patient is much better or if the formula has become negative, the salvarsan need not be given oftener than twice a year.

The serological formula may become normal under ordinary hygienic conditions especially if the patient lives in the open air. This is a clinical fact which should be borne in mind. If the disease is accompanied by evidences of meningeal (exudative) syphilis, the treatments may need to be doubled in intensity.

It is sometimes stated that the iodides are not needed, or do no good in tabes, but this is not the case. After salvarsan and mercurial treatment patients sometimes improve further under the iodide of sodium in moderate or large doses. Indeed forty years ago nearly as much was claimed by good observers for massive doses of iodide as is now claimed for salvarsan.

Along with these first medicinal measures, the physician should prescribe something which is also most important, and that is simply *rest.* Every patient with locomotor ataxia should at once have the importance of rest strongly impressed upon him, and the prescription of sixteen weeks a large part of the time in bed is sometimes advisable. Equally good results can be usually obtained, however, by obliging the patient to go through a simple and regular life, involving moderate walking and no hard physical work. Institutional life for three months is of enormous advantage. It is a rule to which I have seen hardly an exception that tabetic patients brought to the hospital improve in a striking way simply

from the quiet routine of life there, and despite the thinness of city milk and the uncertain value of hospital eggs.

The drugs which are used as tonics are mainly the nitrate of strychnia, iron, the preparations of arsenic and phosphorus. The various preparations of the phosphates, such as glycero-phosphate of lime, the hypophosphite of lime, phosphoric acid, seem to be of some benefit. Strychnine occasionally does good in small doses, but unless very carefully increased large doses may lead to disastrous results and it should always be given with caution. A great many other drugs may be given for the relief of symptoms. Thus ergot, belladonna, and urotropin sometimes help the bladder. The fluid extract of buchu in doses of twenty drops, combined with ten drops of the tincture of hyoscyamus, is also useful. For the pain, phenacetin is a drug which gives satisfaction. It may be combined with bicarbonate of sodium, with codein; or some of the other coal-tar products, such as antipyrin, antifebrin, pyramidon, may be substituted. A teaspoonful of baking soda internally will sometimes stop the pains. Small doses of strychnine may be combined with this. Small amounts of strychnine can always be given for the sexual weakness, but the dose should rarely be made a large one. For the gastric crises, nothing is so good as a hypodermatic injection of morphine, and for the severe crises of pain an occasional hypodermic of morphine should be given. The locomotor ataxic, however, who becomes addicted to the use of morphine for his pains is indeed in a hopeless condition. In persistent constipation the diet should be light and mainly of vegetables and of liquids, such as milk, broths, etc. In persistent diarrhœal states I have found ichthyol of use. This drug also relieves the pains.

The annoying insomnia is to be treated by fresh air, lukewarm baths and sea-shore life. If drugs must be used, bromide of lithium with a few grains of chloral, paraldehyde in not over thirty-drop doses or the veronal group are the best.

In neuralgia of the rectum and bladder, suppositories containing iodoform and belladonna, or codein, or antipyrin, may be used. Sometimes simple gelatin or gluten suppositories act very well. Some of the cases of rectal neuralgia or hyperæsthesia are due to insufficient clearing of the lower bowel when a movement occurs, and if the patient washes out the bowel with a pint of warm water after each movement he is very much more comfortable.

There is no diet which has a specific effect upon locomotor ataxia, but the patient should be given those foods which are non-fermentative and digestible.

Hydrotherapy is of considerable benefit. The most efficient of the single measures is the lukewarm bath at a temperature of about 95°F.

for ten or twenty minutes daily. After the bath it is well to have a little cold water poured over the back and then the patient should be diligently rubbed. In most cases a simple lukewarm bath is quite as effective as anything. In others the patient feels better if there is added to it some slight stimulant to the skin, *e.g.*, two tablespoonfuls of pine-needle extract; or a regular pine-needle bath or Nauheim bath may be given. The Charcot douche given in moderate strength is helpful in cases that are not advanced or particularly weak. I have some hesitation in recommending any special watering-places or cures. I have had patients return benefited from the Hot Springs of Virginia and other American resorts. In Europe, the baths at Lamalou, France, and at Nauheim, Germany, have some reputation. Hot baths are sometimes injurious, and bathing may be overdone by the ataxic.

Electricity is of use from its general tonic and reflex effects, and it perhaps exercises some direct influence on the diseased process. Strong galvanic currents (15 to 30 ma.) should be applied along the spine, through the trunk, and down the legs and arms. The combined galvanic and faradic current is even better, given in the same way. The faradic brush may be applied over the anæsthetic extremities and along the back.

The actual cautery is occasionally efficient in stopping pains. It should be applied to the back as often as twice a month at least and sometimes twice a week. Large dry cups may be applied rapidly and in great number (80 to 100) along the spine and along the course of the sciatic nerves. They make the patient feel better and relieve sometimes the sense of exhaustion. In very painful cases occasional wet cups and leeches are useful.

I still find that suspension by the neck and arms is helpful in some cases. It is best adapted to persons in the second stage and to those who have a good deal of bladder trouble and pain. It is of little value in the paralytic stage, and must be used with care in the early stage and when patients are large and heavy. Suspensions should be given for from one to three minutes three times a week until twenty-five or thirty are taken. After three months a second course may be given.

The treatment of locomotor ataxia by systematic exercises, known as the Frenkel method, is one that has been effectively used. It consists in having the patient go through regular exercises which teach him to co-ordinate the different groups of muscles of the trunk, legs and arms. The Frenkel method is one which can be given with advantage to persons who are passing into the second stage of tabes and in whom the disease is not making progress. It is especially indicated where there is not great hypotonia. It must be used very carefully and for a long time. It often enables the patient to walk better and use his arms better, but it does not especially affect the progress of the disease.

Finally, it has seemed to me that those sufferers from locomotor ataxia do best who persistently and courageously fight against their malady. Those who, despite suffering and discomfort, will three or four times a year take medicinal treatment, and some hydro-therapeutic or electric measures such as will have beneficial effect upon their general nutrition and such as will buoy up their hopes and improve their mental condition, are quite sure to be rewarded. After a hard fight they will emerge into a state of comparative relief from their symptoms and secure a measurable degree of rest from the progress of their disease.

LATERAL SCLEROSIS

(*Spastic Spinal Paralysis*)

Adult Form.—Spastic spinal paralysis or spastic paraplegia is a malady due to disease of the lateral columns and especially the pyramidal

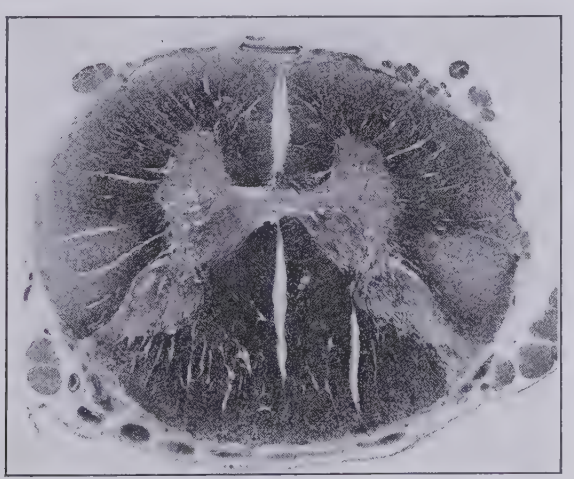


FIG. 126.—Lateral scleroses with spastic paraplegia showing degeneration of pyramidal tracts.

tracts of the spinal cord. As these tracts are diseased and injured in many ways and by many causes, spastic spinal paralysis is a very common condition. It is symptomatic of transverse myelitis, of multiple sclerosis and of tumors and other pathological conditions which cause pressure on the cord. It occurs in the cerebral diplegias.

There is, however, one condition in which in adults the lateral columns and especially the pyramidal tracts undergo primary degeneration, and thus we speak of primary lateral sclerosis, causing a form of spastic paraplegia which is an individual disease (Fig. 126). It is a very rare condition; its existence was long doubted, but it has now been established.

It is a chronic malady characterized by progressive spasticity and weakness of the legs and later of the arms, by increase of the reflexes, the development of contractures and by the absence of sensory disturbances and sphincter troubles. The disease develops between the ages of twenty and forty. The causes are unknown, except that there is one type of the disease which is of hereditary origin. Syphilis, acute infections and toxic substances are sometimes associated with its development. Poisoning by certain kinds of lathyrus cause symptoms of the disease.

In the hereditary or familial form the disease affects different members of many succeeding generations in the family. In the cases so far described it begins early in life, affects only or mainly the legs and runs a very slow course unaccompanied by pain, ataxia or visceral symptoms. Both the acquired and the familial forms may last for twenty or thirty years.

The special symptoms of the disease are the same as those which occur in involvement of the pyramidal tracts from other causes, and are described under the head of myelitis.

In the diagnosis of the disease one has to exclude pathologically the spinal form of multiple sclerosis, chronic transverse myelitis, compression paraplegia from vertebral disease and from tumors. The disease is so rare that particular pains must be taken to rule out these more common disorders.

The Congenital Form (Little's Disease).—*Etiology.*—The term spastic spinal paralysis, is also applied to an affection which is always of congenital or infantile origin and is due, it is supposed, to a lack of development of the pyramidal tracts. This lack of development leads to a sclerosis of the lateral columns of the spinal cord and to symptoms of rigidity of the legs and arms, exaggeration of the reflexes, with some real muscular weakness and atrophy. The disease is of prenatal, natal or infantile origin, being due to some developmental defect or, as Little supposed, to premature and forced deliveries, with probably some meningeal hemorrhage on the convexity of the brain. Through these causes the pyramidal tracts cease to grow, or, at least, this process is greatly delayed. In many cases there is a premature birth and difficult labor.

Symptoms.—The malady is noticed within a short time after birth, usually within a year, but it may be delayed in family types to the fifth year or even later. Some cases of Little's disease may, it is believed, develop as late as after maturity. It is not my purpose to give a description in detail of the symptoms of this trouble, because they are given under the head of cerebral diplegia or birth palsy. The only difference between ordinary cerebral diplegia and the disease under present con-

sideration is that in this latter form there are no marked mental defects; the child is not small headed and idiotic, nor does it have epilepsy or cranial nerve palsy or hydrocephalus. The brain seems to be spared except so far as its motor functions are concerned. It is convenient to separate this type of disease from the ordinary spastic cerebral palsies with mental defect, for the reason that the future of these cases is in some instances more hopeful. As they mature, the lateral columns occasionally gain in development and some increase in the strength and control of the limbs is obtained. I base this statement upon the experience of others. In several cases of Little's disease at the age of fifteen to twenty-five which I have seen, there has been no marked improvement. Mentally, however, these patients are often very bright.[1]

Fig. 127.—Little's disease showing adduction of thighs and cross-legged progression.

Children with this trouble on trying to walk are obliged to cross one leg in front of the other as they are helped along, giving them a characteristic "cross-legged" progression. The arms are less affected than the legs. The facial and throat muscles may be slightly involved. There is no pain. In some cases the disability increases as the child grows older, owing to the greater size and clumsiness of the patient. The arms become much stiffened and contractured, and the hands are flexed so that the patient can neither walk nor help himself. Epilepsy and mental deterioration also may develop at the time of puberty or adolescence but they do not belong to the typical form.

Prognosis.—The mild cases that learn how to walk and can use the arms and hands may grow up, slowly improving, and reach a good age and a fair degree of health. The severer cases rarely reach adolescence, but grow gradually more helpless and generally succumb to some intercurrent disease before they are twenty.

Diagnosis.—The disease is distinguished from ordinary cerebral diplegia (birth palsy) due to brain lesion by the absence of epilepsy, mental defects and microcephalus. (See Cerebral Diplegia.)

[1] It is due to American neurology to say that Dr. Seguin as long ago as 1879 said: "It is possible that tetanoid paraplegia in young children may be due to deficient cerebral development and consequently agenesis of certain tracts of the cord."

From compression myelitis, the involvement of the arms and the absence of pain and disturbance of sphincters are distinctive. Hereditary spastic paraplegia runs in families, begins at the fourth or fifth year, and involves chiefly the legs.

Treatment.—This is altogether one of mechanics and attention to nutrition. The limbs must be persistently *masséd;* tenotomies should be performed so as to straighten the legs; constant voluntary effort to use the stiffened muscles should be made. Resection of certain of the posterior roots has given some favorable results. Braces, roller crutches, etc., should be used. Patience is often greatly rewarded in this disease.

THE COMBINED SCLEROSES

By the combined scleroses is meant those forms of degenerative sclerosis in which both the posterior and lateral columns are involved.

There are several diseases in which combined sclerosis exists. They are:

1. Combined scleroses of profoundly anæmic and toxic states (Putnam's type).
2. Hereditary spinal ataxia (Friedreich's ataxia).
3. Combined scleroses complicating general paresis.
4. Tabetic form.

There are many cases reported in literature of combined scleroses, but the clinical pictures vary very greatly. These cases are probably in the most part forms of chronic myelitis or multiple sclerosis with ascending and descending degeneration. Marie has shown that the vascular supply of the spinal cord is such as rather to favor the development by extension of sclerosis in the lateral and posterior columns from a chronic leptomeningitis, and his suggestion that many of these cases are perhaps of syphilitic origin accords with my experience and conviction. Some years ago, Gowers described a disease that he called ataxic paraplegia, the lesion in which, he believed, lay in the lateral and posterior columns. Most of the cases which belong to this clinical description I think can be properly classed either with the cases of locomotor ataxia, of multiple sclerosis or of some form of chronic myelitis.

THE COMBINED SCLEROSES OF PERNICIOUS ANÆMIA AND CACHECTIC STATES

(*Putnam's Type*)

This form of disease was first described by Dr. J. J. Putnam, later by myself. It is certainly rather common in this country, being almost as frequent as multiple sclerosis.

Etiology.—The disease occurs oftener in men. Eleven out of my twenty-three cases were in women.

The age of most cases is between 50 and 65 (16 out of 23), and next to this between 40 and 50 (5). The youngest case was 36 (Rothmann's). There is often some family or personal neuropathic history. I have reported three cases occurring in one family.

The disease follows acute infections like influenza, prolonged diarrhœal or dysenteric attacks, and serious dietetic errors. One patient had a malignant tumor.

The most important cause is that associated with severe or pernicious anæmia, which in turn is attributed to gastro-intestinal disturbance and consequent toxæmia. Probably some intestinal micro-organism is behind the whole. Syphilis is not a cause. Often, if not always, however, nervous symptoms develop before evidences of extreme or extended anæmia are present.

Symptoms.—The initial nervous symptom is nearly always a persistent paræsthesia of the feet and hands associated with some weakness of the part. The paræsthesia is of the stocking or glovetype and gradually ascends the legs and arms. It is rather sharply limited. The tip of the tongue and nose may be affected. Following this there is a good deal of ataxia, with some loss of deep sensibility, and this symptom increases with the loss of motor power. Tactile, thermic and pain anæsthesiæ are not marked until late in the disease, but the patient often has severe pains in the back and limbs and often complains bitterly of his parasthesiæ. There may be some differentiation of sensibility as in syringomyelia. With the weakness of the legs there is at first an increase of knee-jerks, and sometimes ankle clonus and rigidity, so that the patient shows the symptoms of ataxic paraplegia. Later the spasticity may become less and the knee-jerks disappear.

When the arms are involved, paræsthesia, weakness, awkwardness and a very slight anæsthesia develop and progress. The mind may be slightly weakened, the memory becoming defective and the patient emotional. The cranial nerves are not often involved, though optic atrophy has been once noted. The blood presents evidences of anæmia. Usually this is secondary, but in about 40 per cent. of cases there are the typical findings of pernicious anæmia. It should be noted that a large proportion of cases of pernicious anæmia have slight involvement of the spinal cord and show some acro-paræsthesia and motor weakness, but do not develop the disease.

The disease progresses rather rapidly, though hopeful remissions of six to twelve months occur. In from six months to a year, if there is no remission, the process has nearly reached its height. During this time the patient emaciates, marked anæmia or pernicious anæmia sets in. The skin is sallow and pale, and pigmentation is seen. Diarrhœa occurs at times.

The bladder becomes weak and retention of urine follows; the control of the rectum is eventually lost, and the patient lies bed-ridden with paralysis and contractures of the lower limbs. The arms do not become so badly paralyzed.

Prognosis.—Death usually occurs in about two years in the progressive cases. It may be prolonged to three years, and I have found that if the trouble is recognized early the process may be checked. The prognosis is better in older patients.

Pathology.—The disease is a toxic one, but whether due to the failure of some glandular tissue of the body to act or to infection or poison is not known. As the disease belongs to the degenerative period of life, it may fairly be supposed that in some cases the blood-making organs undergo premature decay and that the nerve centres are poisoned in consequence;

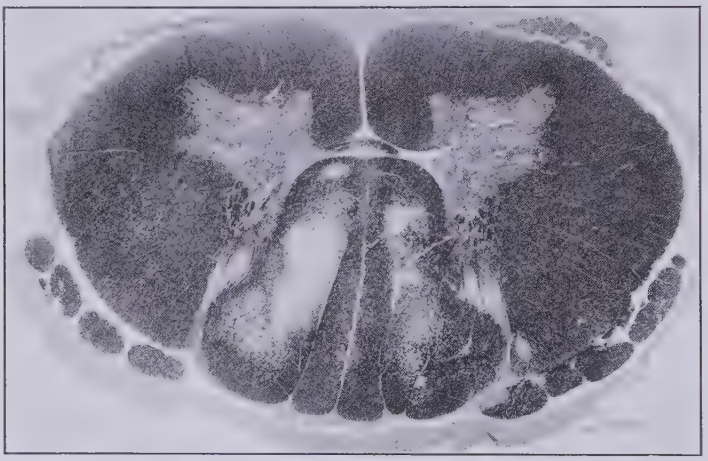


FIG. 128.—Lesion of posterior column in pernicious anæmia.

or the intestinal tract may be the origin of the poison since in the severe anæmias there is great atrophy of the mucous membranes. The process is classed as chronic degenerative myelitis by some writers, but I see no reason for regarding it as a true inflammation.

As to the location of the trouble, the posterior columns of the spinal cord are the parts first and most involved (Fig. 128). The process especially involves the postero-median parts. It appears to start in certain foci in the lower dorsal or cervical cord, and to spread thence. New foci apparently develop as the disease progresses; the lateral columns are also severely affected, and especially the crossed pyramidal tracts (Fig. 130). The other columns are involved, but less regularly and completely. The lower dorsal and lower cervical levels of the cord are especially selected. The anterior horns are finally involved, but the change

here comes later. Finally, there may be actual softenings in the cord, producing cavities (Fig. 130). Sometimes the pathological process is irregularly diffuse and not systemic. The blood-vessels are somewhat affected with hyaline degeneration, but I am unable to say how important a factor this is. The walls are thickened; there are in places dilatations and hemorrhages, and in other places collapsed vessels. There is no evidence of inflammatory reaction. The meninges are not involved, nor are the anterior and posterior roots much affected. Examinations of the peripheral nerves (Rothmann) and of the brain have been negative.

Diagnosis.—The disease is easily distinguished from locomotor ataxia by the absence of syphilitic history, the rapid onset, the anæmia, the peculiar progressive acro-paræsthesia, the motor weakness, the absence of ocular symptoms and of lightning pains. There is usually also at first an exaggeration of the reflexes. The steady and rapid progress of the malady is also most characteristic.

Multiple neuritis is excluded by the slow onset, the marked ataxia, the absence of muscular wasting and of tenderness and pain, and the development of bladder and rectal symptoms.

One of my cases presented such a marked differentiation of cutaneous sensations that I believed it to be one of syringomyelia. The necropsy showed a cavity in the cervical cord due to softening, and in a measure explained, if it did not justify, the diagnosis. The steady and rapid progress of the symptoms ought to exclude such a diagnosis.

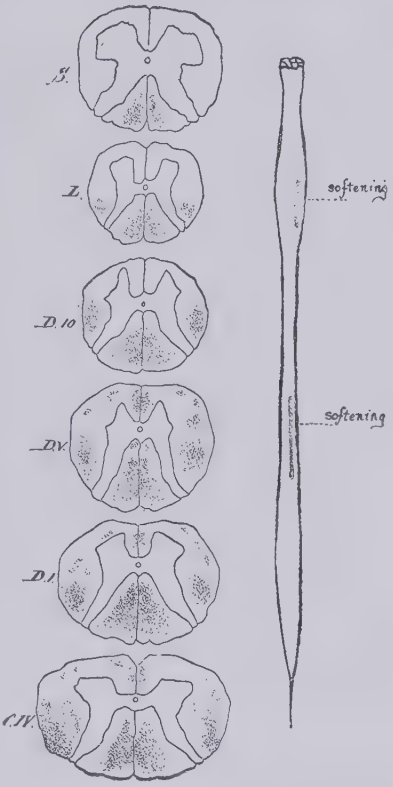


FIG. 129.—Combined sclerosis of toxic origin, showing location of degeneration of original foci of disease.

The existence of marked anæmia, and especially of pernicious anæmia, is, taken with acro-paræsthesia, ataxia, paralysis and rapid course, pathognomonic.

The essential nature of this process is a parenchymatous degeneration, affecting apparently the axon-tissue first. The same poison which causes pernicious anæmia does the work here, only it may affect the nerv-

ous centres before causing a true essential anæmia. It seems even as if in some cases the neural change came first.

Treatment.—The use of arsenic sometimes is helpful and brings on remissions in those cases associated with anæmia. Intestinal antiseptics and colon washings, abundant food, salvarsan injections, transfusions, fresh air and tonic medication are the measures indicated.

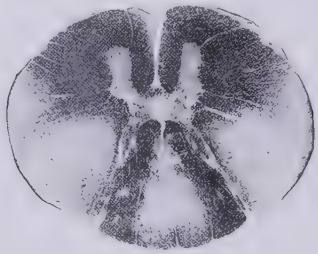


FIG. 130.—The spinal cord in combined sclerosis from pernicious anæmia, dorsal region. The columns of Goll, crossed pyramidal tracts, and cerebellar tracts are affected.

HEREDITARY SPINAL ATAXIA

(Friedreich's Ataxia)

There are three forms of hereditary or congenital spinal and cerebellar defects which should be grouped together: They are: (1) Hereditary spinal ataxia, or Friedreich's ataxia: (2) hereditary cerebellar ataxia; and (3) hereditary spastic paraplegia. They are quite similar in cause and mode of development. The difference in symptom depends upon the fact that the defect exists in the one case mainly in the posterior and lateral columns of the cord in the second in the cerebellum, and in the third mainly in the lateral columns.

Friedreich's ataxia, the most common of all the forms, is a chronic degenerative disease mainly affecting the posterior and lateral columns of the cord.

Clinically, the disease is characterized by ataxia beginning in the lower limbs and gradually involving the upper limbs and the organs of speech. Curvature of the spine, talipes, and finally paralysis and contractures appear.

Etiology.—The fundamental factor in predisposition is an inherited or connate lack of development of the spinal cord, more particularly of the posterior columns and pyramidal tracts. This condition is inherited directly sometimes, but indirectly as a rule; that is to say, the parents or other members of the family usually show simply a neurotic history, and it is in only a minority of cases that there is a history of ataxia in the direct line of ancestry.

The more frequent condition is this: the parents or grandparents have some neuroses, such as insanity, inebriety or great nervous irritability; then the ataxia occurs in the children of the next generation. Sometimes in a single family the uncles and nephews or cousins may be found to have the disease. Hence the name "family ataxia," used by some writers. There are a good many cases in which the parents were apparently perfectly sound and healthy. The parents rarely have locomotor ataxia, though this has been observed in a few cases.

Syphilis in the ascendants is an element in some, probably in many cases. Habitual intemperance in parents is perhaps sometimes a factor; much more rarely consanguinity and tuberculosis act as predisposing causes of degeneration. More cases have been observed in America than in any other country; while the fewest have been reported from France.

The disease develops at about the time of puberty, most cases occurring between the ages of six and fifteen years. It is not very rare, however, for symptoms to develop even in infancy, though some of the cases reported at this time were probably of a syphilitic character.

In a given family the disease, as a rule, strikes the older members first, but the younger members are attacked at a relatively earlier age. The most typical time of development is a rather late one; *i.e.*, after twelve years of age. The disease may come on after maturity. In American cases the age of development of the disease has been rather earlier than the average.

The male sex slightly predominates, its proportion being about 60 per cent. In America the female sex has, however, been more affected (3 to 2).

The patients, as a rule are the children of the laboring and agricultural classes. They have been found in the country oftener than in crowded cities. The families have often been large, but this is not always the case, especially in American cases. Usually the disease appears after infectious fevers, such as diphtheria, variola and typhoid.

Symptoms.—The child is first noticed to have an uncertainty in the gait and some feebleness in the lower limbs. These symptoms gradually increase until they interfere seriously with progression, and force him in two or three years to leave off active play. With this there may be some slight pains or numbness in the lower limbs, and an examination will show, within a year or earlier, that the knee-jerk is gone. After five or six years the arms become affected with inco-ordination, and a little later bulbar symptoms, such as thick or scanning speech, and often nystagmus, appear. These bulbar symptoms may be very much delayed. During this time the patient suffers little pain or anæsthesia and has no trouble with the bladder or rectum. Vertigo and headache are often

present. Dorsal flexion of the toes, talipes varus or some other form of clubfoot, and lateral curvature of the spine are often observed (Fig. 131). Oscillation of the head and choreiform or inco-ordinate movements of the extremities may develop. As the disease progresses the legs become weaker, and finally paraplegia, with contractures and muscular wasting, sets in. The intelligence is sometimes diminished. The disease makes slow progress; often it remains almost at a standstill for years, and the patients usually die of some intercurrent disease, such as phthisis or an infectious fever.

Among the rarely observed symptoms are tremor of the hands and head, spasms, sluggish pupils, tachycardia, profuse sweats, impotence, slight vesical incontinence, fragilitas ossium. Many of these symptoms occur only late in the disease. When there is marked involvement of the cranial nerves, the disease may be put down as a cerebellar-spinal trouble.

Fig. 131.—Friedreich's ataxia showing late deformities of feet.

The major and essential symptoms are: (1) ataxia, beginning in the lower limbs and extending to the arms and tongue; peculiar rolling, ataxic gait; (2) disturbances of speech; (3) talipes and spinal curvatures; (4) gradual development of paraplegia; (5) loss of knee-jerk; (6) absence of cutaneous anæsthesia, of bladder troubles, of eye troubles except nystagmus, and of severe pains; (7) the development of the foregoing at about the time of puberty.

Diagnosis.—The disease must be distinguished from multiple sclerosis of spinal type, and from cerebellar ataxia and tumors. The family history is usually of help. The disease begins earlier in life than does multiple sclerosis. The speech disturbances, nystagmus, paræsthesiæ, pallor of the optic discs, intention tremor, and exaggerated reflexes also serve to distinguish multiple sclerosis. The differentiation from hereditary cerebellar ataxia is not important as the two diseases are really the same. The general symptoms of brain tumor are not present in cerebellar ataxia.

Pathology.—The lesions of importance are found in the spinal cord and medulla only. The cord is usually small, flattened and apparently congenitally imperfect in development. In some cases two central canals have been seen. A sclerosis exists throughout the whole length

of the posterior and lateral columns, sometimes extending to the anterior columns (Fig. 133). The sclerosis is most marked in the postero-median columns, which are eventually affected *in toto*. The postero-external column is less involved and there is often a narrow strip of healthy tissue between the posterior horn and the sclerosed area, also between the posterior gray commissure and the diseased parts. The posterior-column sclerosis is usually most marked in the lumbar region. In the lateral columns the sclerosis always affects most the crossed pyramidal tracts. The direct cerebellar tracts and the so-called ascending antero-lateral tract are diseased in some cases, but apparently not in all. In a few instances the anterior median columns are involved. A zone of healthy tissue is often found between the sclerosed pyramidal tracts and the posterior horn. There are no important changes in the gray matter except a secondary atrophic process. Some chronic leptomeningitis, especially on the posterior surface, has been noted. The medulla shows some traces

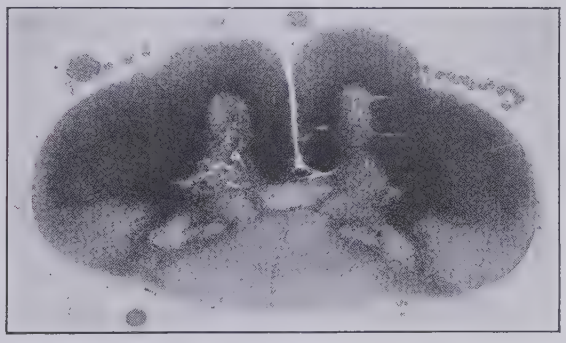


FIG. 132.—Friedreich's ataxia.

of extension of the sclerosis, but the involvement of the cells of the hypoglossal nucleus is probably the most significant change. The brain exhibits no changes of importance in relation to the symptomatology of the disease. The posterior nerve-roots are extensively sclerosed, the anterior roots less so, and the peripheral nerves show some degenerative changes. The peripheral nerves are much less involved than in tabes dorsalis. It is asserted that the sclerosis in the cord is really a neuroglia proliferation—a gliosis—and there is no doubt a large amount of neuroglia proliferation in the diseased areas.

Course and Prognosis.—The disease is a progressive one, though it may be stationary for a long time and may even show temporary improvement. The longest period of duration of the disease on record is forty-six years and the shortest two years, the average being fifteen or twenty years. Death occurs from some intercurrent disorder or from bulbar complications.

Treatment.—A quiet life, good food and favorable hygienic surroundings are the main therapeutic helps. Arsenic and various nerve tonics

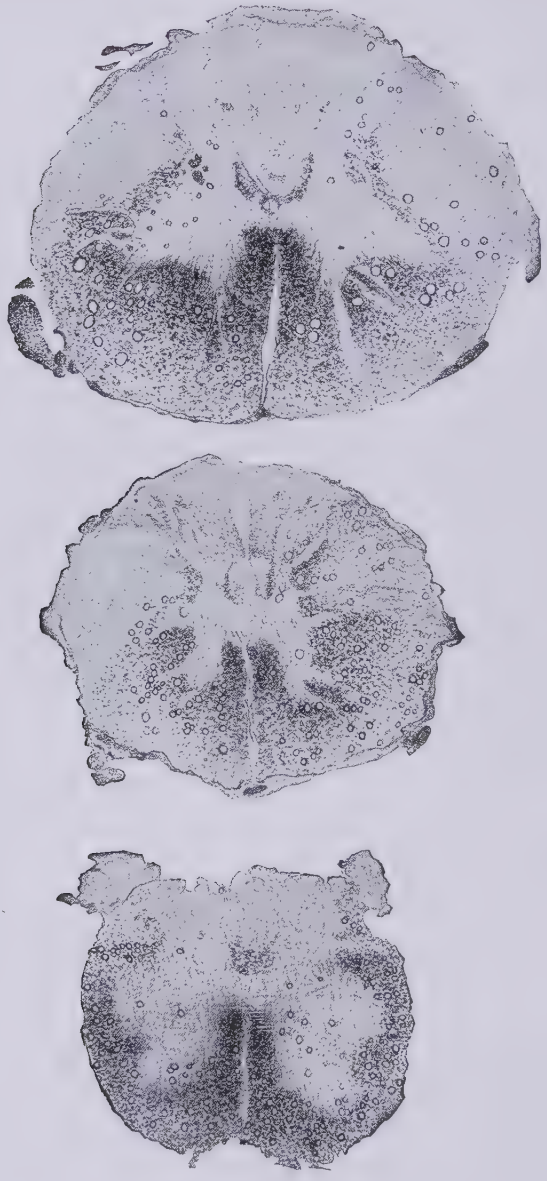


Fig 133.--Spinal cord in a long-standing case of hereditary ataxia.

may be of temporary benefit. If blood tests give evidence of syphilis in the patient or parent, salvarsan, mercury and iodide are indicated.

A course of mercurial injections or inunctions is advisable in the early stages.

Hereditary Spastic Paraplegia is an extremely rare malady resembling in general course the adult form of lateral sclerosis (p. 279).

HEREDITARY CEREBELLAR ATAXIA

This is a disease so akin to hereditary ataxia that some writers think it useless to try and distinguish them. I can only say that in my experience the disease sometimes shows itself mostly in cerebellar and cranial nerve symptoms.

The first symptom is shown in a disturbance of the gait. This is indicated by clumsiness and stumbling and by a tendency to rolling and pitching like a drunken man. There are inco-ordination and jerkiness in the movements of the arms and sometimes choreic movements. The patient also has oscillation and jerky movements of the head. When lying down the inco-ordination is very much lessened. The speech is hesitating, ataxic and explosive. The eyes show jerky movements somewhat like those of nystagmus. An important symptom in some cases is the development of optic neuritis followed but not always by optic atrophy and blindness. The knee-jerks are usually exaggerated. The patient has no anæsthesia and suffers little pain, although he may have some headache. There is no disturbance of the sphincters. Mentally, the patients are usually somewhat deficient, becoming eventually either simply childish or actually demented. The malady progresses and ends like that of the spinal type.

Pathological Anatomy.—The few autopsies which have been made show an atrophy of the cerebellum. In some cases this is macroscopic, the cerebellum being reduced to one-half or one-third its size. In other cases there is no naked-eye change, but there is found microscopically an atrophy of the cortical matter.

Diagnosis.—In the cerebellar type, the distinguishing features are the presence of the knee-jerks, the absence or late occurrence only of a spastic or ataxic paralysis, the presence of speech defects, of nystagmus and jerky tremors and of the group of other special cerebellar symptoms; also the occurrence at times of an optic atrophy. In the spinal form the early and more prominent paraplegic symptoms develop, with deformities of the feet, while there is a late involvement only of the cranial nerves.

CHAPTER XIV

THE PROGRESSIVE MUSCULAR ATROPHIES AND MUSCULAR DYSTROPHIES

The peripheral motor neurons of the spinal cord, and their terminal end organs, the muscles, form a trophic unit, and the same degenerative disease may attack either end or any part of this physiological mechanism. There is a clinical and pathological relationship between the different spinal and muscular types of atrophies. But there are sufficient differences, also, as to oblige us for convenience sake to make certain classifications. Thus those disorders which attack chiefly and first the anterior horn cells are called *progressive muscular atrophies;* those disorders attacking first the muscle tissue and its nerves are called *progressive muscular dystrophies.* The progressive muscular atrophies of central origin may attack the motor-nerve cells of the eye, of the throat and lips, of the upper or lower spinal cord. In accordance with the level affected, the disease has received different names. Sometimes the pyramidal tracts of the spinal cord are first and most involved, and later the peripheral motor neuron is affected. This has furnished excuse for another type. Then, again, while most cases of muscular atrophy are acquired, there are types which are hereditary. Thus we find the disease classified as follows:

Progressive muscular atrophies.	Spinal form.	Duchenne-Aran or common type. Luetic type. Hereditary types: Infantile. Adult. Charcot-Marie-Tooth.
	Bulbar form.	Progressive bulbar paralysis.
	Mesencephalic form.	Progressive ophthalmoplegia.
	Upper neuron form	Amyotrophic lateral sclerosis.

The progressive muscular dystrophies have also been much subdivided, but they are essentially the same disease, as will be seen later.

Of the above progressive atrophies, I have already described ophthalmoplegia (page 100).

PROGRESSIVE MUSCULAR ATROPHY

(Progressive Spinal Amyotrophy; Duchenne-Aran's Disease)

This is a disease characterized by a slow, progressive atrophy of the muscles of the extremities and trunk, with consequent paralysis, not accompanied by any notable sensory disturbance, and due to a progressive atrophy of the lower motor neurons in the spinal cord. In the more typical and common form (Duchenne-Aran's) the disease begins in the upper extremity.

Etiology.—The disease affects persons in the middle period of life (twenty-five to forty-five). The extremes are fourteen and seventy years. It is more frequent in males. Heredity is rarely, if ever, a direct factor. Great mental strain, exposure, traumatism, excessive use

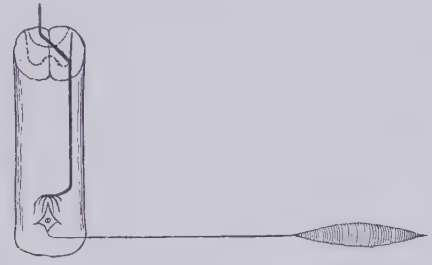


FIG. 134.—Showing: 1. Segment of spinal cord with motor neuron, end brush, and lateral tracts, the parts affected in progressive muscular atrophies; and 2, the muscle and its nerves, the parts affected in progressive muscular dystrophies.

of certain groups of muscles, acute infectious diseases—especially typhoid—childbirth, acute rheumatism, and lead poisoning are among the causes. All three types of atrophy may be caused by syphilis, and hence the disease may occur in locomotor ataxia. Usually, however, syphilis causes an atrophy running a rather special course.[1]

[1] Among seventy-two cases, four-fifths were in males. Spyhilis was present in about one-fifth of the cases. With regard to age, I have seen two cases beginning as early as the twelfth year, of the Duchenne-Aran variety, in the most characteristic form. In one case, which I have now watched from the time it began, at the twelfth year, to the present time, the patient now being twenty-four, the disease was arrested after about four years of progress, the patient being left with simply an atrophy of the left forearm, in the muscles of the ulnar distribution. My oldest patient was one of a perfectly typical arm type of atrophy seen with Dr. J. Arthur Booth, occurring in a physician aged seventy-two, but beginning, probably, before his seventieth year. The period during which the greatest number of cases were seen was that between the thirtieth and fortieth years, and next, between that of forty and fifty. In cases of bulbar paralysis the age of fifty-three seems to be a singularly serious year. I note the occurrence of six cases at about that period of time, among twelve in all. There were about five times as many cases among males as among females, but this does not apply to all types of the disease. In the bulbar cases there were more women, the proportion being 8 to 4.

Among the exciting causes, by far the most frequent is that of some form of strenuous occupation. Workmen who had to do very heavy work, athletes or professional

Symptoms.—The patient suffers at first from slight rheumatoid pains in the shoulder or arm, associated with some feelings of numbness and weariness. Muscular wasting then begins to appear in the intrinsic muscles of one hand. The adductor longus pollicis is very early affected as are the thenar muscles and the interossei. The atrophy spreads from muscle to muscle, and does not follow the distribution of nerves, although the ulnar-nerve supply is most seriously disordered. The ball of the thumb becomes flattened, and the patient cannot abduct or flex it well. When the radial interossei are reached the forefinger cannot be abducted, and this is often an early sign. The disease gradually extends upward, attacking the flexors more than the extensors of the wrist, then the upper arm and shoulder, affecting abduction and flexion of forearm especially. Meanwhile the hand has become thin and flattened, flexion of wrist and extension of fingers are lost (owing to the more special involvement of the ulnar nerve) and a characteristic "griffin-claw" appearance results. After a time (three to nine months) the other arm begins to be affected. Occasionally there is a temporary remission.

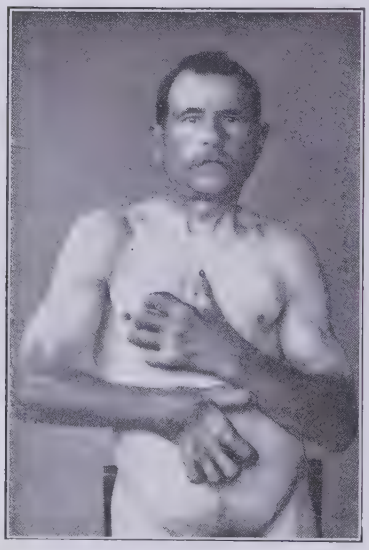


FIG. 135.—Showing wasting of hands in muscular atrophy.

In a few cases the atrophy begins first in the shoulders and arms, attacking the deltoid, biceps and triceps, then extending downward to the hands. This constitutes the "upper-arm type."

If, as is usually the case, the disease continues to progress, it passes from the shoulder girdle to the deep muscles of the back, then downward, involving successively the thigh muscles, the glutei, the crural extensors and abductors being oftenest chosen. The leg muscles may be finally involved, but they usually escape. The disease as it descends continues its progress in the trunk, involving the intercostals. It slowly as-

contortionists were among the victims, and in the cases of bulbar paralysis, there was several times a history of the patients being very great and excessive talkers. Lead, as a cause of paralysis, does not seem to me, after all, as important a factor as has been supposed. It could be blamed for the disease in not over five cases, and not certainly in all of these. Several of my patients were very heavy users of tobacco, but I fancy this was only a coincidence. Two patients had in infancy a previous attack of poliomyelitis. In one case there was a very distinct history of an acute infection by dengue; it is not improbable that other cases have followed an acute infection. On the whole the dominant causes are an occupation strain, syphilis, and some inherited weakness of the affected neurones.

cends the neck also, and finally leads to a paralysis of the diaphragm, or a bulbar palsy may set in.

It will be seen that the ordinary course of the disease is from the lower-arm muscle groups (ulnar and median) up to the shoulder group (middle cervical nerves), then down through the dorsal and lumbar nerves, rarely reaching the sacral groups, then up. In very rare cases it begins in the legs and ascends.

Along with the wasting there are a corresponding weakness and paralysis, but the paralysis is the result of the atrophy and does not precede it. Fibrillary twitchings of the muscles occur; the idiopathic muscular contraction caused by striking it a blow is often marked; myoid tumors are easily brought out. In some cases the muscles are flaccid and toneless, and the deep reflexes, knee-jerk, and arm-jerk disappear early (atonic atrophy), but in most cases the irritability and tonicity of the muscles are increased, the knee-jerks exaggerated, and we have tonic atrophy. This condition may be so marked as to make it resemble a special clinical type of progressive atrophy known as *amyotrophic lateral sclerosis*.

The electrical irritability of the muscles gradually lessens to both galvanic and faradic currents, but no marked qualitative changes occur at first. Eventually we may get partial degeneration reactions, but these occur late in the disease, unless this runs a very rapid course, when fairly typical degeneration reactions may be got early.

In typical cases of progressive muscular atrophy there is no anæsthesia, cutaneous or deep, and when such symptoms develop the presence of peripheral disease or of syringomyelia or spinal tumor must be suspected. The patients may suffer from rheumatic-like pains and from paræsthesias.

The affected parts often show excessive sweating and congestion and evidence of vasomotor disturbance. This may involve the face on one or both sides; one pupil may be larger than the other, due to irritation of the cilio-spinal centre. The iris reflex, however, is preserved, and the optic nerve is never involved.

The sexual power is often weakened, but the sphincters are not attacked. The urine shows variations in the amount of urea. There is usually an increase of lime salts.

Complications.—The most common complication is an extension of the process to the medulla, causing disturbance of speech and swallowing (bulbar paralysis). Muscular atrophy complicates locomotor ataxia, but is rarely complicated by it. An apparently typical and flaccid atrophy may develop spasticity and signs of involvement of the pyramidal tracts; in other words, it may turn out to be an upper neuron type or amyotrophic lateral sclerosis.

Course and Duration.—The disease usually progresses steadily until it has reached an advanced stage, when it may stop. Remissions may occur early, however, and even some improvement take place; the disease then ordinarily progresses again. It lasts from two years to thirty or more, but on the average not over ten or twelve years. Death usually occurs from pulmonary disease, owing to the weakness of the respiratory muscles. Sometimes the extension to the medulla and involvement of the muscles of deglutition and of the larynx are the cause of death.

Pathology.—The primary anatomical change is a degenerative atrophy of the neurons of the central parts and anterior horns of the gray matter of the spinal cord. The atrophy gradually extends and involves the whole anterior horn. It also extends vertically, first down, then up. Consecutive to this there is atrophy of the anterior roots, peripheral nerves and the muscles. The disease begins in the deeper parts of the anterior cornua, involving the central and median groups of cells. These are more concerned in nutrition and in the finer muscular movements of the extremities. Hence atrophy always precedes or at least keeps pace with paralysis. The levels affected are the lower cervical and upper dorsal; but if the disease is extensive the dorsal, lumbar and sacral cord are also involved. The affected part is nearly free from nerve-cells, and those present are atrophied, their processes are short or absent, and the cell has lost its angular appearance. Sclerotic and pigmentary changes are observed. The neuroglia and connective-tissue cells are increased in number, but there are no marked changes in the blood-vessels, though these may be much dilated. There is often some degeneration of the lateral columns. It is confined chiefly to the pyramidal tracts, but extends somewhat anteriorly into the mixed lateral column. It does not affect the cerebellar or ascending lateral tracts. The anterior columns may be slightly affected. The posterior horns, columns and roots are normal.

The affected muscles show various degrees of degeneration. They are pale and streaked with yellow, due to fatty deposits. Some fibres may be simply narrow and shrunken; others have lost their striation and become granular from deposit of fat globules or degenerated muscle elements; other fibres have lost their striations and appear as if filled with a homogeneous, glassy-looking substance containing a few fat granules (vitreous degeneration); others show a longitudinal striation. The interstitial connective tissue is increased and in places has taken the place entirely of the muscles. The capillaries and small vessels are distended. Healthy fibres may be seen among the diseased. Changes have been found in the sympathetic nervous system, but they are unimportant.

The diagnosis has to be made from the progressive muscular dys-

trophies, chronic poliomyelitis anterior, syringomyelia, neuritis and neuritic family atrophy.

In the muscular dystrophies there is commonly a history of heredity; the disease begins usually in childhood or adolescence. It attacks the lower limbs oftener; it is slower in progress and the two sides are affected at the same time; there are no fibrillary contractions, and the degeneration reaction does not occur till very late.

Chronic spinal atrophy or chronic (subacute) anterior poliomyelitis is usually due to syphilis. It begins rather suddenly and, having reached its height, does not progress, but remains stationary or improves for a time. The paralysis occurs first, the wasting follows. It affects groups of muscles physiologically related more strictly than does progressive muscular atrophy. Lumbar puncture gives positive evidence of syphilis with a formula like that of tabes.

Syringomyelia is distinguished by the presence of peculiar sensory and trophic disorders.

Neuritis caused by lead poisoning is detected by the history of the case, its tendency to affect the extensors of the arm chiefly, and the absence of a progressive tendency. Sometimes, however, lead poisoning and palsy end in a progressive muscular atrophy.

Ordinary multiple neuritis is distinguished easily by its rapid onset and the presence of sensory symptoms.

The hereditary or neuritic type of progressive muscular atrophy is characterized by its attacking first and mainly the legs and forearms, by the presence of a good deal of sensory disturbance, of typical degeneration reactions, and of a hereditary or family history.

Treatment.—The patient should be well fed and have rest, quiet and fresh air. Careful local faradization and galvanization of the spine and affected parts are indicated. Massage does no good, but very carefully applied active and passive exercises are useful.

Hypodermatic injections of strychnine in the affected member, gr. $\frac{1}{80}$ to $\frac{1}{30}$ daily, the internal use of arsenic, phosphorus, iron, quinine and cod-liver oil sometimes are beneficial.

In cases with a syphilitic history, specific measures are indicated and injections of salvarsan should be given to the toxic limit.

It is necessary to admit that we have no remedy for non-luetic types of this disease.

THE LUETIC TYPE OF SPINAL ATROPHY

(Remittent Type of Chronic Anterior Poliomyelitis)

This form of atrophy runs a course like that of a chronic anterior poliomyelitis and it is described under the head of myelitis, p. 243.

HEREDITARY TYPES OF SPINAL AND NEURO-SPINAL ATROPHY

These are: (1) the infantile form (Werding-Hoffman), (2) the adult form (described by the author), (3) the Charcot-Marie-Tooth type.

1. In the infantile form the disease begins in the first or second years of life. It attacks first the muscles of the pelvic girdle, the back, buttocks, hips. It then goes to the shoulders and neck muscles and finally involves the forearms, legs, hands and feet. The atrophy is symmetrical and at first is concealed by subcutaneous fat. Paralysis and atrophy go along together. There are no fibrillary twitchings. The paralysis is flaccid and the deep reflexes disappear. Scoliosis and leg deformities occur. There are no sensory symptoms.

2. The adult form, *familial type of progressive spinal atrophy*, attacks the patient in the third or fourth decade of life. It begins in the thighs or arms but soon extends to the legs and forearms. The paralysis seems to precede the atrophy. There are some fibrillary twitchings, and degenerative electrical reactions are present. The paralysis is flaccid, deep reflexes disappear. The disease runs a relatively rapid course. In six months the patient is unable to walk; the arms are affected and later the bulbar muscles. The patient dies within a year.

No exciting cause is known. The serological formula is negative, so that the disease is not a manifestation of syphilis.

HEREDITARY MUSCULAR ATROPHY OF PERONEAL TYPE

(Charcot-Marie-Tooth Type)

This is a hereditary or family muscular atrophy of central (and neuritic) origin, attacking the legs and later the forearms. The symptomatology of the disease has had so many accessions that its clinical features are much obscured. I follow the descriptions of Marie and Lèri, and my own personal observation of several cases.

The disease is quite rare. It affects males more than females, but the difference is not great. It almost always begins before the age of twenty. It attacks first usually, the muscles of the leg, not the foot, involving the peronei, causing a varo-equinus or a flail foot; then calf muscles, the extensors of the toes and the intrinsic muscles of the foot are involved. The thighs escape till later. After some time the forearms and small hand muscles are reached. The shoulders and arm, neck and trunk muscles escape. There are occasionally fibrillary contractions, and always partial or complete degenerative electrical reactions. The patients complain of some pain and numbness, but there is no anæsthesia.

The characteristic, then, of this form of atrophy is that it affects the legs, rarely going above the knee, and producing a rather symmetrical

atrophy of these limbs, so that in the later stages the legs look very spindle-like, and there is no pseudo-hypertrophy at any period in the progress. In some cases the hands and forearms are attacked in a similar way, producing a symmetrical and progressive atrophy of the parts. Gowers has observed a case in which with the characteristic involvement of the forearms there was also an involvement of the face, in a fashion somewhat similar to that seen in the facio-scapulo-humeral type of dystrophy.

The disease runs a long course, with remissions, and resembles in prognosis the dystrophies.

Some authorities assert that the disease is due to a progressive degenerative neuritis. In the writer's opinion, the anterior horns of the spinal cord are primarily attacked, a view recently confirmed by Marinesco. It is therefore a disease of the peripheral motor neurons.

The *treatment* is the same as for the other forms of hereditary muscular atrophy.

FIG. 136.—Peroneal type of atrophy. (*Marie.*)

A disease apparently related to the above is progressive hypertrophic interstitial neuritis (Déjerine and Sottas). This also affects first and mainly the legs and feet and runs a course like that of the Charcot-Marie disease. It is characterized by progressive atrophy, by a palpable hypertrophy of the nerves, kypho-scoliosis and bulbar symptoms. It is usually of hereditary and familial origin and begins in infancy or adolescence.

GLOSSO-LABIO-LARYNGEAL PARALYSIS

(Progressive Bulbar Paralysis)

This is a disease characterized by progressive wasting and paralysis of the muscles of the tongue, lips, palate and throat, due to an atrophy of the nuclei of the nerves supplying those parts.

Etiology.—It is a disease of the degenerative period of life, most cases occurring after forty and between that time and seventy.

The disease begins later in life than spinal atrophy. It occurs rather oftener in men than women. A neurotic heredity is sometimes noted. Exposure to cold and excessive use of the muscles in talking, mental strain, debilitating influences, lead and syphilis are causal factors.

The element of syphilis in producing this type of atrophy has been studied by the writer, and he has found that in over 20 per cent. there is a history of infection. It has occurred in two cases as a terminal symptom in locomotor ataxia. Dr. Janeway tells me of a case in which there was a prompt remission by the use of salvarsan.

Symptoms.—The tongue is the part first affected, and the patient finds that he speaks indistinctly and cannot especially articulate the lingual consonants *l*, *r*, *n* and *t*. The tongue movements become weak. It cannot be elevated and is protruded only a little distance. It gradually atrophies and looks scarred and wrinkled. The lips become weak and the patient cannot whistle nor make the consonants *p*, *b*, *m*, or the vowel *o*. The saliva begins to dribble from the mouth. Disturbance in swallowing soon develops. Hard solids are taken with difficulty, next fluids, while semisolids are generally managed best. The lips finally become so paralyzed that the mouth cannot be shut, and the lower part of the face is motionless and expressionless. The upper face wears an expression of anxiety and suffering, the saliva dribbles constantly and the whole physiognomy of the patient becomes characteristic and pitiful in the extreme. The facial nerve may get somewhat involved. Articulation becomes almost entirely lost; the voice has a nasal twang from paralysis of the palate.

The patient has tired and uncomfortable sensations of dryness and stiffness about the throat. There is no pain or anæsthesia, but occasionally there is impairment of the sense of taste. The throat reflex is usually lost, so that tickling it causes no reaction.

Electric irritability is at first unchanged, but in the later stages partial degeneration reaction occurs. In rare cases there is a rapid pulse and still more rarely glycosuria.

The laryngeal reflex becomes weak; the adductor muscles act feebly also, but abductor paralysis is rare.

The mind is not affected, but there are often an emotional weakness and tendency to tears—not at all unreasonable in view of the distressing nature of the malady.

The disease is often the terminal stage of spinal muscular atrophy; it may be associated with the latter, with amyotrophic lateral sclerosis, or with ophthalmoplegia. All these types may occur together.

It runs a progressive course, with remissions of a few weeks or months. It lasts from two to three or four years.

The termination is eventually fatal except perhaps in luetic cases. Death occurs through interference with swallowing, and inanition or a broncho-pneumonia or bronchitis may develop which ends the patient's life.

Pathology.—The primary lesion is found in the nuclei of origin of the hypoglossal, glossopharyngeal, vagus and spinal accessory nerves. The raphe fibres and the anterior pyramids are also usually somewhat involved. There is sometimes atrophy of the cells of the facial nerve and of the nucleus ambiguus, which is the motor nucleus of the vagus. The brunt of the disease falls, therefore, upon those more superficial

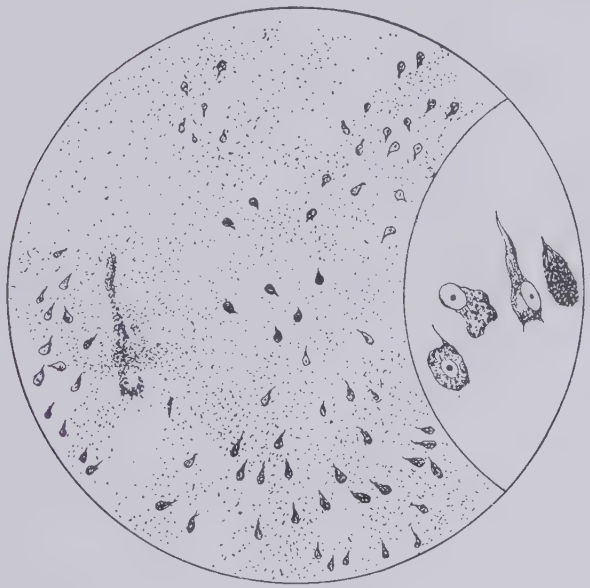


FIG. 137.—Bulbar palsy; nucleus of hypoglossal. The dark cells are nearly obliterated with pigment, ×2–3. To the right are four cells in different stages of degeneration, ×1–6.

or posterior nuclei which are representative of a continuation of the anterior cornual cells. If the disease is complicated with amyotrophic lateral sclerosis, or progressive muscular atrophy, or ophthalmoplegia, we find atrophy in the pyramidal tracts, cord and ocular nuclei. The atrophic process is similar to that observed in the spinal disease.

The muscles of the tongue, and to a less extent the orbicularis oris and the throat muscles, show evidences of degeneration and atrophy. In some cases the tongue is not shrivelled, owing to the presence of a fatty deposit, and on account of this the disease has been divided into atrophic and paralytic types, but this distinction is unnecessary.

Diagnosis.—The disease must be distinguished from polioencephalitis inferior, bulbar apoplexy, tumors, and softening, from multiple sclerosis, and from chronic lesions of the cerebral hemispheres causing pseudo-bulbar paralysis. It must also be distinguished from myasthenic bulbar palsy. The slow onset, the progressive course, the bilateral character, the absence of involvement of sensory nerves, and the degenerative reactions are sufficient for a diagnosis. In myasthenic bulbar palsy there is progressive paralysis, but none of the typical atrophy of the parts; the ocular muscles are usually involved; often these patients get well. It is important always to note whether there are ophthalmoplegia and spinal muscular atrophy associated with the disease.

Treatment.—The patient should be kept quiet, and he should be overfed. The same drug treatment as in the spinal disease is indicated. Small doses of morphine, gr. $\frac{1}{24}$ to $\frac{1}{36}$, and of atropin may be given also. It is important in these cases, where there is any suspicion of syphilis, to give salvarsan and a course of hypodermatic injections of mercury. Massage to the neck and face gives temporary relief. Careful voluntary exercises of the weak parts are helpful. Electricity should be tried for a short time twice or even thrice daily, if possible. The faradic current may be used, alternating or combined with the galvanic. Galvanization of the neck and medulla does no good. After a time it may be necessary to feed with a tube or even to do tracheotomy.

INFANTILE HEREDITARY BULBAR PALSY

There have been reported cases of progressive bulbar palsy beginning in infancy and of hereditary or family type. They are often associated with spinal atrophy and amyotrophic lateral sclerosis. Some of these cases are probably muscular dystrophies.

MYASTHENIA GRAVIS

(*Asthenic Bulbar Paralysis and Asthenic Bulbospinal Paralysis*)

These names are given to a chronic frequently remitting disorder characterized by the symptoms of mesencephalic and bulbar paralysis or by the symptoms of this condition and of progressive spinal paralysis, the distinguishing features being that there is no muscular atrophy, that the cases often continue on for many years instead of going on to a fatal issue, and also by the fact that on autopsy no surely characteristic microscopical changes are found.

Etiology.—Little is known as to the cause of the disease. The majority of cases have been under the age of thirty, but a patient of my own was over fifty years of age and another over forty. It is some-

times associated with anæmia and also with intestinal toxæmia. Recently it has been found that there are lymphoid deposits in the muscles, and in some cases the thymus is present and diseased. Those causes which are found in progressive bulbar and spinal paralysis, viz., overwork, mental strain, are sometimes found here.

Symptoms.—The disease usually begins gradually and oftenest affects first the muscles of the throat and face and of the eyes. The patient notices a general feeling of unnatural weakness, and gets more easily tired at his work. Then he finds that sometimes he sees double, or one of his lids droops and it is difficult to keep the eye open; then he has to give up chewing solid food because it tires the jaws. Speech soon tires him; the voice may be nasal; and he has to swallow with some

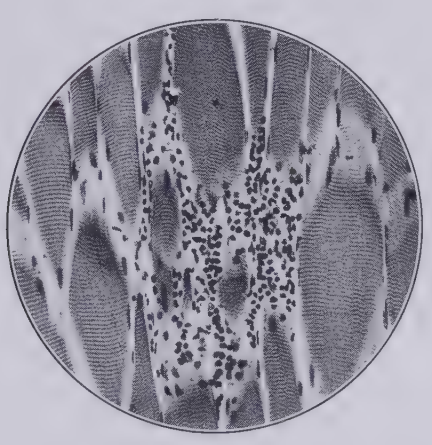


Fig. 138.—Lymphorrhage in muscle in myasthenia. (*Dr. F. S. Mandlebaum.*)

care. The arms get easily weak and they tire at their accustomed tasks. Digestion is slow and constipation may occur. The abdominal muscles relax and the abdomen is distended.

The mental and other bodily functions are little disturbed. There is little or no pain, no emaciation or local atrophy; no spasm or twitching. On stimulating the muscles with an electric current the responses get weaker and finally cease. After a rest they return (myasthenic, electrical reaction). The same phenomenon occurs on tapping the tendons of affected muscles. The symptoms are characterized by remissions; after a patient has reached a point at which he is almost moribund, he begins to get stronger again and may slowly get into a state of comparative strength; then the symptoms slowly return. In this way the disease may continue for a number of years. The patient sometimes dies of exhaustion, but he often recovers if properly treated.

Pathological Anatomy.—In the half-dozen early autopsies no lesion of the nervous system was found, except microscopical changes in the cell of the motor nuclei. Later lymphoid-cellular deposits (lymphorrhages) in the muscles and a persistent or diseased thymus have been found. Most careful examinations of the blood, urine, gastric and intestinal contents have been made for me by Dr. T. W. Hastings but have failed to throw light on the disease, which is due, probably, to a toxæmia of intestinal or blood-gland origin.

Diagnosis.—The clinical characteristics which distinguish this disease from progressive muscular atrophy and true bulbar palsy are the transient diplopia and ptosis, the absence of any true atrophy of the muscles of the face or tongue or extremities, the absence of fibrillary twitchings, the presence of the myasthenic reaction and the irregular course with remissions. Like these other diseases, however, asthenic paralysis is not accompanied by any disturbance of sensibility or any impairment of the sphincters.

The treatment consists in complete rest, careful attention to feeding, and the use of iron, arsenic, strychnia, the calcium salts and intestinal disinfectants. In some cases excellent results are obtained by injections of massive doses of strychnia, up to gr. $\frac{1}{4}$ ter die hypodermically. By one or other of these measures all patients if seen reasonably early can be relieved or cured.

AMYOTROPHIC LATERAL SCLEROSIS

(Spastic Form of Progressive Muscular Atrophy)

This disease is one which has the closest possible kinship to progressive muscular atrophy. Its clinical symptoms, however, are somewhat different, and anatomically there is a somewhat more extensive and peculiar change. Amyotrophic lateral sclerosis, or Charcot's disease, is characterized by progressive spasticity with atrophy, rigidity and contractures of the limbs, ending or beginning often with bulbar symptoms.

Etiology.—It is a rarer disease than progressive muscular atrophy, and occurs most often between the ages of thirty and fifty, involving the second part of adult life. Rare cases, however, have been reported as occurring in childhood. The female sex is rather more often affected. No definite exciting cause is known. It is sometimes due to syphilis. It is, probably, fundamentally a disease of involution; *i.e.*, a teratological defect, the first and second motor neurons degenerating because of inherently deficient vitality. This state of affairs, however, underlies the other spinal myopathies also.

Symptoms.—*Spinal Onset.*—In its spinal and more common form there is first noticed a weakness and clumsiness of one hand and this may be associated with some subjective sensation like a numbness. No atrophy

is seen but some twitchings of the muscles, and irregular jerky tremor of the fingers and hand occur. Soon the other hand and then the legs are involved, so that in a few months walking tires the patient, going upstairs is difficult, the feet tend to drop because the anterior tibials are weak. Cramps are noticed in the leg muscles, and the same paræsthesiæ are felt as in the hands and arms. In less acute cases the lower limbs are not involved for a long time; *i.e.*, for one or two years. Examination after the disease has developed shows some slight atrophy of the intrinsic hand muscles and perhaps of the forearms. The fibrillary twitchings are present and sometimes are extremely marked affecting the shoulder and trunk muscles as well as the arms. The reflexes are much exaggerated, especially those of the lower extremities; there is ankle clonus and the dorsal flexion of the great toe (Babinski's sign) is present. The wrist, elbow and perhaps the jaw-jerk are also very active.

The disease usually progresses steadily, until the upper extremities become almost useless and the lower extremities nearly as much so. Atrophy has now set in, of the same type as that of progressive muscular atrophy. No special sensory symptoms except sense of weariness and aching are to be noted and there is no involvement of the sphincters. Finally, in about one and one-half to two years the medulla becomes affected, and there is weakness, stiffness, cramp and atrophy of the tongue; the lips are less mobile and speech and swallowing are affected, the symptoms being the same as in bulbar palsy except that there is evidence of a rigidity and a tendency to cramp and contracture, while the atrophy comes later.

Bulbar Onset.—Sometimes the disease begins in the medulla. There is then, on the whole, a more rapid progress, a later atrophy and a slighter tendency to remission than in the other myelopathic atrophies. The patient first notices some difficulty in speaking or swallowing. He feels at times a spasmodic drawing of the tongue or stiffness of the cheek or lips. Soon after there appear a weakness and stiffness of the legs and arms. The symptoms progress rather slowly. The speech becomes disturbed; swallowing is difficult; the arms atrophy and become stiff and rigid, producing characteristic deformities. There is great exaggeration of the reflexes; the legs show the presence of ankle clonus; all the arm reflexes are increased, and the jaw is stiff and has a very lively jerk when struck. The patient suffers little from pain. There are no anæsthesias and no sphincter trouble, except in the last stages of the disease.

Pathological Anatomy.—Post-mortem examinations show a very marked sclerosis involving the direct and crossed pyramidal tracts; also some of the short-fibre systems of the lateral column. The anterior cornual cells are atrophied, as in progressive muscular atrophy. Lesions are also seen at times in the columns of Goll. In fact, the post-mortem

findings resemble entirely those of progressive muscular atrophy, except that there is a sharper accentuation of the disease in the lateral tracts. In the medulla the nuclei of the hypoglossal and other motor cranial nerves will be found diseased and the pyramidal tracts also. The lesion of the white columns diminishes in intensity from below up, so that as one gets into the cerebral peduncles very little if any is to be seen. In a few cases, however, the process has been traced to the motor cortex and changes even in that part have been discovered. In a case of my own, which was very closely studied and reported upon by Dr. Joseph Collins, the sclerosis of the motor tracts did not reach above the medulla, and there was no lesion of any moment in the cortical motor cells.

In amyotrophic lateral sclerosis the degenerative process attacks first the terminal fibres and collaterals of the cortico-spinal motor

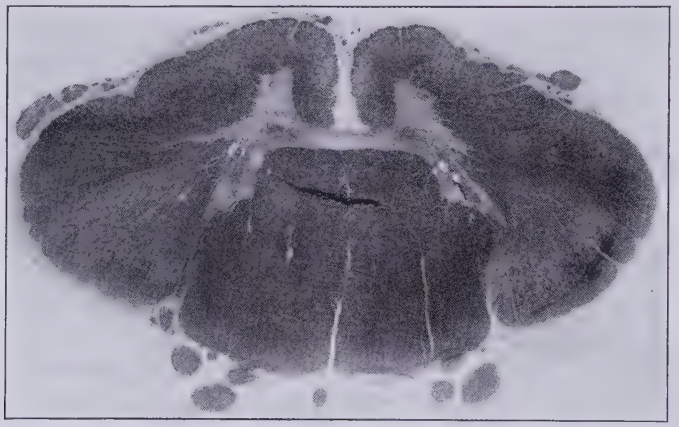


Fig. 139.—Amyotrophic lateral sclerosis. Shrunken anterior horns, lateral sclerosis.

neurons. The next part attacked is the anterior cornual cell. We have, therefore, the curious and perplexing phenomenon of a disease which attacks the cell body of one neuron and the terminal neuraxon of another neuron just above it. It is difficult to explain this upon the ordinary lines of nerve-cell pathology. Still, we have analogies, perhaps, both in locomotor ataxia and in multiple neuritis.

The diagnosis of amyotrophic lateral sclerosis must be made from transverse myelitis, multiple sclerosis, and the other forms of progressive muscular atrophy. The diagnosis is based upon the very striking and progressive atrophy associated with myokymia, exaggerated reflexes, rigidity and contractures, without any sensory symptoms or sphincter troubles. The diagnosis from ordinary bulbar palsy depends upon the appearance of stiffness, cramps, exaggerated reflexes and rigidity displayed in the muscular supply of the facial, the trigeminal and

the glossopharyngeal, and the tenth, eleventh and twelfth cranial nerves.

The **prognosis** is invariably bad, but in those types beginning in the legs and arms life may be prolonged a number of years.

The **treatment** is the same as that for progressive muscular atrophy.

THE PROGRESSIVE MUSCULAR DYSTROPHIES

As I have already stated, there are various forms of progressive muscular atrophy to which the special name of "dystrophy" is given, because they are hereditary in character and because the muscular end of the motor neuron is apparently the first and the most severely attacked. Recent and closer study of the pathology of muscular dystrophy tends to show that the lesion is not in the muscle and terminal of the motor nerves alone, but that the peripheral motor neuron is also to some extent affected. The clinical characteristics of the muscular dystrophies, however, are pretty distinct and are sufficient to justify the separation of them into a different class.

The general characteristics of the dystrophies as distinguished from the spinal atrophies are the following:

1. There is generally a hereditary or family history.
2. They begin rather early in life.
3. The progress is very slow with sometimes long remissions.
4. The limbs are affected symmetrically, *i.e.*, both shoulders or both legs at about the same time.
5. The tendency is for the disease to attack the root or proximal segments of the extremities of the body, *i.e.*, the hips and the pelvic girdle or the arms and the shoulder-girdle first, and later the peripheral segments of the limbs.
6. There occurs occasionally pseudo-muscular hypertrophy of some muscles.
7. There is occasionally along with the atrophy myotonus of some muscles.
8. There is no idio-muscular reaction.
9. There are no fibrillary twitchings.
10. The deep reflexes disappear *pari passu* with the atrophy of the muscles.
11. The electrical reactions show no degeneration change until very late.
12. There occur certain fibro-muscular contractures causing deformities of the body.

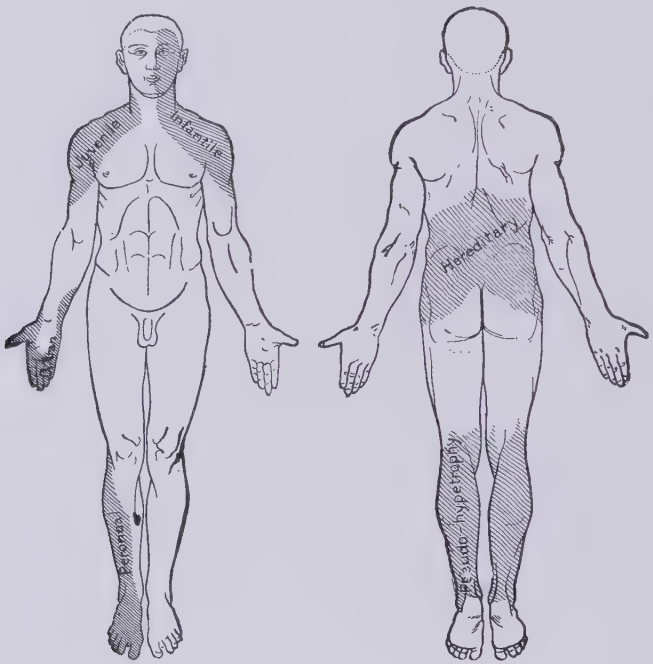
A number of types have been described, the distinctions being based chiefly on the part of the body first affected. These types are not of

great importance, but may be enumerated here for convenience. (Fig. 140):

1. Pseudo-muscular hypertrophy. Allied to it is:

(*a*) Leyden-Möbius or hereditary type, appearing in children, beginning in the back and pelvic girdle and lower limbs. The congenital type of Hoffman is also allied to this.

2. Erb's juvenile type, or scapulo-humeral type, beginning in childhood or youth, usually in the shoulder-girdle and trunk.



A. B.

FIG. 140.—Showing the parts first attacked in the different types of muscular dystrophy and muscular atrophy. The shaded parts in A show the place of onset of progressive muscular atrophy of ordinary or Duchenne-Aran type, and of types 2 and 3 in text. B shows place of onset of types 1 and (*a*) in text.

3. Landouzy-Dejerine type, or infantile progressive muscular atrophy of Duchenne, or facio-scapulo-humeral type. It resembles the preceding form, with the exception that it involves also the face.

The peroneal or leg type has been classed with the dystrophies, but is probably of myelo-neural origin, and has been described with the spinal atrophies (see page 296).

The essential unity of all these different forms is shown by the fact that cases occur in which pseudo-hypertrophy takes place in the scapulo-humeral and other types, by the fact that a disease resembling pseudo-

hypertrophic paralysis occurs without any hypertrophy, and by the fact that different types occur in the same family.

The dystrophies are, in a considerable proportion, of hereditary origin or, at least, run in families. Among twenty-nine cases observed by myself, there was a distinct family history in 12 per cent. They affect boys much oftener than girls, in the proportion of about one to five. In my own cases, there were twenty-four males and five females. The great majority occur under the age of twenty, and a majority under the age of ten years. Thus, among my cases, there were under five years, eleven; under ten years, seventeen; and under twenty years, there were twenty-two. They may develop, however, as late as the fortieth year.

The so-called "peroneal type" develops, on the whole, rather late, while pseudo-muscular hypertrophy and the allied hereditary types begin very early, often in infancy, and some children have even never known what it was to walk.

The distribution of the types probably differs somewhat in different countries and races. In this country, in the Montefiore Home, where the patients are mainly Hebrews, the pseudo-muscular hypertrophy and the hereditary type of Leyden-Möbius is, by all odds, the most common. In the personal cases, seen by myself, both in private practice and in my clinic, pseudo-muscular hypertrophy is also most frequent. The shoulder-arm type of Erb comes next in frequency, while the facio-scapulo-humeral type is extremely rare, there being only one in thirty cases. The peroneal type is not common. Among the native Americans the dystrophies often develop later in life, take a longer and less serious course, and sometimes become arrested. In fact, the common type among the native classes, which I have seen, is one in which the pelvic girdle, back and shoulder girdle become gradually affected rather symmetrically, so that the patient becomes greatly wasted, but is still able to walk and use the forearms, the face and throat not being affected. These cases form the living skeletons of the museums. This form of dystrophy begins usually below and ascends, being first a hip-girdle and trunk affection, then a shoulder-girdle affection, and is, therefore, a sort of combination of the hereditary (Leyden-Möbius) and of the so-called *juvenile* type (Erb).

All the cases which have come under my observation in New York could be grouped, then, in the following way:

1. The typical cases of pseudo-muscular hypertrophy, beginning in the leg, but soon involving the hip-girdle and shoulder.

2. The root type, beginning in the hips usually and back muscles, and extending to the shoulder and trunk muscles, not necessarily ex-

tending to the legs and arms, at least, not until very late in the disease, and accompanied often with some pseudo-hypertrophy.

3. The peroneal type which, in its pure form, is extremely rare, but of which irregular types are not so uncommon.

4. The facio-scapulo-humeral type, which is the rarest of all.

Among my cases there were of the first type, eleven; of the second type, twelve; of the third type, four; and of the fifth type, one.

Fig. 141.—Pseudo-muscular hypertrophy.

PSEUDO-MUSCULAR HYPERTROPHY

(*Atrophia Musculorum Lipomatosa*)

This is a disease beginning in childhood and characterized by a symmetrical, progressive atrophy of muscles, associated with an apparent muscular hypertrophy due to a deposit of fat in the wasting muscles.

Etiology.—The disease attacks boys much oftener than girls. It begins, in the vast majority of cases, under the age of ten, often at the close of infancy, very rarely not till after puberty. Heredity is a very important factor (in three-fifths of the cases), the hereditary influence being almost always transmitted by the mother. A psychopathic or neuropathic condition is often found in the ancestry. Syphilis, intemperance, consanguinity, are not factors in hereditary causation. Injury and an acute disease sometimes appear to act as exciting causes.

Symptoms.—The first symptom noticed is a weakness in the legs, which shows itself in a peculiar "waddling gait" and a tendency to stumble and fall. A little later (fifth or sixth year) an apparent hypertrophy of the leg muscles, particularly of those of the calves, develops. The extensors of the knee or one of them and the gluteal and lumbar muscles may also be affected. Sometimes the hypertrophy is very great, at other times it is barely noticeable. The affected part has a peculiar, hard, non-elastic feeling to the hand, not like that of normal muscle. In the upper part of the body the hypertrophy oftenest attacks the infraspinatus, the supraspinatus and the deltoid (Fig. 141). The lower parts of

the pectoralis major and latissimus dorsi are also usually atrophied, giving a characteristic appearance to the shoulders. The upper-arm muscles usually become wasted, the forearm, neck, and face rarely. The tongue may be hypertrophied.

Along with the pseudo-hypertrophy there occurs an atrophy of certain groups of muscles, and after a time the pseudo-hypertrophy disappears and atrophy takes its place. In the lower limbs the muscles most atrophied are the flexors of the hips, then the extensors of the knee and those of the hip. The calf muscles fail before the anterior tibial. The atrophy and consequent weakness of the lower-limb muscles cause great difficulty in going upstairs, the gait becomes more waddling, and the patient loses the power of getting up when lying on the floor or he climbs

FIG. 142.—Pseudo-muscular hypertrophy, patient "climbing up the legs."

up his legs (Fig. 142). These peculiarities are due chiefly to the weakness in the extensors of the knees, the extensors of the hip, the flexors of the hip and the erector spinæ. By reason of the same defects, the child when standing has an antero-posterior curvature of the spine with the concavity backward (lordosis) (Fig. 141). This is due to the weakness of the extensors of the hips, which, acting from the hips, are unable to tilt the pelvis back. On sitting, this lordosis disappears and is replaced often by a curve in the opposite direction due to weakness of the erectors of the spine. There may be some lateral curvature also. In consequence of the weakness and contractures of the leg muscles, there early develops a talipes equinus, and later the legs may become flexed on the hips and the forearms on the arms.

The muscles show no fibrillary twitching and rarely any degenerative

reactions, but there is sometimes a peculiar tetanic contraction with both the faradic and the galvanic current. A *myotonia occasionally develops on the basis of a dystrophy.*

The knee-jerks and elbow-jerks gradually weaken and in time are lost.

There is no pain or other disturbance of sensibility.

The affected parts feel cold and look reddened, as if from deficient vasomotor innervation. The organic spinal centres are not involved. Intelligence is usually good.

Course.—The disease runs a chronic but variable course. Its progress is at first slow, and after walking becomes impossible it may cease to progress. It lasts from ten to twenty-five years. In a few cases patients have reached the age of fifty or sixty years, even when the disease began in youth. The earlier the disease begins the more rapidly it extends; the more pronounced the tendency to lipomatosis, the more rapid is the course.

Fig. 143.—Leyden-Moebius type of dystrophy. (*Schœnborn and Krieger.*)

Pathological Anatomy.—The disease, like other forms of dystrophy, is a degenerative atrophy, the process affecting first the muscle fibres and nerve terminals, the connective tissue being secondarily involved. In a simple atrophy of muscles, such as follows disuse, the muscle fibres grow smaller and gradually break up and disappear. In degenerative atrophy, the process is accompanied by evidences of irritation, such as swelling of the muscle fibre, proliferation of muscle nuclei, splitting of the fibre longitudinally, and connective tissue proliferation. All these phenomena are seen in the pathological process which takes place in the dystrophic muscles. All the varied changes may be noted in the same muscle. In the early stages there is a true hypertrophy of some of the fibres, a condition thought to be characteristic of the muscular dystrophies in distinction from the spinal atrophies. Besides swelling and hypertrophy of fibres, one sees atrophy of the fibres; the bundles are rounded; there are increase of muscle nuclei, splitting of fibres, vacuolization, and a tendency to break up into fibrillæ (Erb). The connective tissue at first shows evidence of irritation and proliferation. Finally, as the muscular atrophy progresses, connective tissue increases and takes its place, until a dense, hard myosclerosis results (Figs. 145, 146). In some

parts there is deposit of fat in the connective-tissue cells, and this may increase until an extensive lipomatosis exists. In the later stage of the disease the fat deposits are absorbed and there are only atrophied muscle

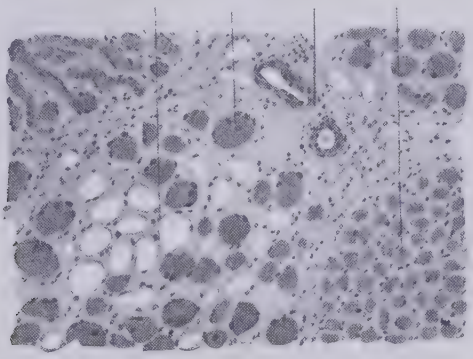


FIG. 144.—Cross-section of crural muscle in dystrophy, showing hypertrophied and atrophied fibres, increase of nuclei. (*Cestan and Lejonne.*)

and connective tissue. The nerves and spinal cord are usually normal; when changes are found they are secondary to the muscular disease.

The process is then, first, hypertrophy of muscle fibre and increase of muscle nuclei, swelling and rounding of fibres, and splitting of the same;

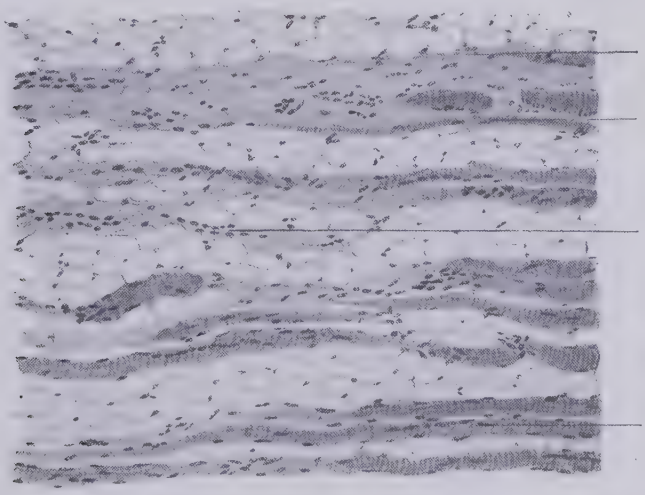


FIG. 145.—Longitudinal section of supinator longus in dystrophy showing atrophic fibres and fat. (*Cestan and Lejonne.*)

then increase of connective tissue, with corresponding atrophy of muscle and deposit of fat.

The process is a primary degeneration due to an inherent nutri-

tional weakness of the muscle. In a measure it is true that those muscles embryologically latest developed are earliest attacked.

The juvenile dystrophy of Erb, or scapulo-humeral form of dystrophy (root dystrophy), begins in childhood or early youth, a little later than pseudo-hypertrophy. The shoulder-girdle is first affected, later the arm. The forearm and legs are attacked very late. Part of the pectorals, part of the trapezii, latissimus dorsi, rhomboid, upper-arm muscles and supinators are affected, while the supra- and infraspinati and forearm and hand usually escape. There may be true and false muscular hypertrophy. There are no fibrillary contractions or degenerative reactions (Fig. 146).

Fig. 146.—Juvenile type of progressive muscular dystrophy; sixth year of disease.

As the disease progresses it gradually affects the hip-girdle and trunk, and finally the patients have an almost general atrophy. This is then the root and trunk dystrophy, which I find the common one in this country.

The facio-scapulo-humeral form, or infantile progressive muscular atrophy begins in early childhood (third, or fourth year) usually, but may develop late. The atrophy attacks first the face, giving a characteristic appearance known as the "myopathic face." There is a weakness of the oral muscle, which causes the lips to protrude and produces a symptom called the "tapir mouth." The atrophy respects the eye muscles as well as those of mastication and deglutition. It extends to the shoulders and arms next, then it pursues the ordinary course of the dystrophies.

Prognosis.—The patient never recovers, but the disease sometimes comes to a standstill and there may even be some improvement, especially in cases beginning late.

Treatment.—The prophylaxis is important. It consists in preventing the marriage of women belonging to dystrophic families; if a

dystrophy has developed in one child, it would be unwise to take the risk of bringing others into the world. Or if children are already born, they should receive the most careful nourishment, outdoor life should be secured, and the dangers from trauma and the infective diseases be prevented. Infants should not be suckled by the mother if she belongs to the dystrophic family.

The moderate use of massage and gymnastics is very important and useful. All kinds of tonic measures are indicated, such as cold baths, good nourishment, arsenic, strychnine, and phosphorus and fats. Tenotomy and other orthopædic measures may be useful in the later stages. Feeding with thymus or other glands does no good.

Amyotonia congenita or *myatonia congenita* is a condition described by Oppenheim and later by Spiller and others. It occurs in childhood and is associated with great weakness, flaccidity and atony of the muscles of the lower and sometimes of the upper extremity. The limbs may be moved like flails; the muscles are not apparently wasted, but react poorly to electrical stimulation. The reflexes are weakened or lost. Active movements are feeble but not entirely lost.

The disease is congenital and in some cases may be the result of accident of intra-uterine life. There is no history of heredity. In mild cases the disease seems to be a purely muscular one; in severer cases the central nervous system is involved. So it is still a question as to whether it should be classed among the muscular dystrophies.

UNILATERAL HYPERTROPHY OF THE FACE

Dr. D. W. Montgomery has collected nine cases of this affection, seven being congenital and one commencing in the second year of life. One followed a neuralgia of the fifth nerve. Dr. Montgomery reported a case in which the development began at the age of ten years. In this case, the hypertrophy involved the bone, and it seems to have been a type of leontiasis ossea.

HEMIHYPERTROPHY

There have been reported in literature by Möbius, Demme, and C. B. Tilanus a few cases of hypertrophy of one side of the body. In one case, the child was born with a half-sided muscular hypertrophy. In the other cases, the disease seems to have been also of congenital origin. The trouble seems to be a kind of teratological defect, and not due to any progressive disorder of the muscular system.

SUMMARY OF THE HEREDITARY OR FAMILY NERVOUS DISEASES

The student may well be confused by the large number of so-called family nervous diseases which modern neurology has discovered and

differentiated. The practical importance of them all is, perhaps, slight, for they are extremely rare, yet it is necessary that they be recognized and properly distinguished, for the prognosis and degree of suffering differ very much in different cases. They are all chracterized by the fact that they are found in different generations and in different collateral branches of a given family, and that they are not necessarily or often passed on directly from one parent to another. The list which I append contains the important types.

Hereditary Chorea.—This is really a kind of hereditary paresis or brain softening. It does not develop until adult life, as a rule, and patients with it may live until middle age.

Hereditary amaurotic idiocy is a family disease, early described by Dr. Sachs, characterized by a general degeneration of all the nerve-cells of the central nervous system associated with blindness and a peculiar degeneration of the optic nerves.

Hereditary Cerebral Diplegia.—This is a family disease in which children between the ages of one and five develop spastic paralysis and sometimes imbecility.

Hereditary Hemiplegia.—This is a family disease which has been referred to by Hoffmann and of which I have seen two cases in my clinic. The children are born hemiplegic and there is atrophy of the hemiplegic side, but in my cases there was no mental defect or epilepsy.

Hereditary cerebellar ataxia is a disease allied to Friedreich's ataxia, but developing somewhat later; *i.e.*, about the time of puberty.

Hereditary spinal ataxia, or Freidreich's disease, is fully described elsewhere.

Hereditary Spastic Spinal Paralysis.—Spastic paralysis in very rare cases is found to run in families, affecting different members of many succeeding generations. In the cases described, it begins at about the age of five, affects only or mainly the legs, runs a very slow course, is not accompanied by pain, ataxia or visceral symptoms; and runs a course lasting twenty or thirty years.

Hereditary progressive Spinal Muscular Atrophy.—There is (*a*) an infantile form (Werding-Hoffmann type) and (*b*) a form beginning in in middle life or later, described by the writer.

Hereditary Muscular Atrophy of Peroneal Type (*Charcot-Marie Tooth*).—This has been described under the spinal atrophies.

Hereditary progressive dystrophies include a large number of types as above described.

ARTHRITIC MUSCULAR ATROPHY

In inflammation of joints the muscles moving them are affected by a simple atrophy which is called arthritic.

Etiology.—Rheumatic arthritis is the commonest cause.

Symptoms.—The shoulder-girdle muscles are oftenest affected, next the muscles of the thighs and legs. Whatever the joint, it is the extensor muscles which are first and most attacked, while the muscles above the joint are more susceptible to the atrophy than those below. The atrophy is rather rapid in the first few weeks and then becomes slower. The muscles affected waste throughout their whole length. They show no fibrillary contractions and no degenerative electrical reaction. There is often an increased irritability, so that an exaggerated tendon reflex or even clonus may be produced. There is no pain or tenderness or anæsthesia in the muscles.

Pathology.—The anatomical change is a simple atrophy and shrinking of the muscle fibres, with some increase in muscle nuclei, little vacuolation, no swelling or splitting of fibres (Darkschewitch). There is some increase in interstitial tissue, but this is slight. The nerves and spinal cord are normal.

The atrophy is probably due in part to disuse and in part it is a reflex trophic disturbance.

The *prognosis* is good. If the arthritis gets well the muscles are also restored.

The treatment consists of electricity and gentle massage, exercise and internal remedies directed to the arthritis.

OCCUPATION MUSCULAR ATROPHIES

As a result of constant over-use, muscles sometimes atrophy. This applies especially to the smaller muscles of the hand. Thus there occurs an atrophy of the thenar eminence in lapidaries and in persons who constantly use this group of small muscles.

Typical atrophies of this kind may be seen in persons who run elevators and who have constantly to grasp the rope in one hand; in golfers who use a special grip; in artisans who have to use excessive pressure on motor nerves. The biceps sometimes wastes in smiths and the calf muscles in ballet dancers. In most cases this occupation atrophy of muscles reaches a certain stage and stops. If the patient is given rest, recovery takes place. This is especially true if the patient is young and in vigorous health. In other instances, the simple occupation atrophy will actually pass over into progressive muscular atrophy. The trouble is usually due to a local neuritis caused by trauma.

CHAPTER XV

TUMORS AND CAVITIES OF THE SPINAL CORD

Frequency and Cause.—Tumors of the spinal cord occur about one-half as often as tumors of the brain. Among 1,000 nervous cases there will be an average of one or two spinal tumors. Men are affected oftener than women. The susceptible age is that of middle and active life. Children do not often have spinal tumors and old people have only the metastatic and secondary type. Injuries to the back are sometimes the exciting cause. Naturally, the presence in the system of cancer, sarcoma, tuberculosis and Hodgkin's disease is a predisposing cause of deposits in the cord.

Location.—Tumors of the spinal cord are:

Extra-dural.	1. Vertebro-spinal.	Cancer. Sarcoma.
	2. Spinal.	Sarcoma, fibroma. Neuroma, lipomata.
Intra-dural.	3. Extra-medullary.	Lipoma, neuroma. Fibroma, sarcoma. Tubercle, cysts, etc.
	4. Intra-medullary.	Endothelioma. Sarcoma. Glioma. Cancer, etc.

All parts of the spinal cord are attacked, the cervical region, however, is most frequently involved and next in nearly the same frequency are the conus and cauda equina. Then come the lower part of the dorsal cord, the upper part of the dorsal cord and last of all the lumbar and upper sacral cord. The figures representing the proportional distribution would be somewhat as follows:

Cerical	7	Upper dorsal	3½
Conus and cauda	6	Lumbar	2
Lower dorsal	5		

The relative frequency of the different forms of tumors is shown in the following table of personal cases of vertebro-spinal and spinal tumors:

Sarcoma	24	Tuberculoma	2
Carcinoma	12	Cyst	2
Glioma	9	Neuro-fibroma	2
Endothelioma	4	Adenoma	1

If one leaves out from this table the vertebro-spinal tumors the relative frequency would be about the same, except for the carcinomata.

The most frequent form of tumor, as is seen above, is the sarcoma; next to this the carcinoma, glioma and endothelioma. The sarcoma undergoes various changes and is sometimes osteosarcoma, sometimes fibrosarcoma, gliosarcoma or endosarcoma. The endothelioma is a common tumor or not, apparently, according to the laboratory in which it is examined. A number of sarcomata are considered by some authorities to be endotheliomata.

Tuberculoma is relatively rare and syphiloma is a tumor that I have never seen and has been rarely reported in this country.

If one groups glioma (which is the cellular type of glioma proliferation) with syringomyelia (which is a fibril stage of proliferation) we would probably find that the most frequent form of new growth which attacks the spinal cord is gliomatosis in some form.

Cysts and fibromata, parasites and adenoma are rare and almost curiosities in the pathological laboratories of neurologists.

VERTEBRO-SPINAL TUMORS

Nature and Frequency.—The tumors of the vertebral column, which later involve the cord, are carcinoma, and less often sarcoma or osteosarcoma. Carcinoma is almost invariably of metastatic origin, the primary source being oftenest the breast, less often the stomach, uterus and prostate. Sarcomatous tumors may be primary. Other forms of vertebro-spinal tumor are very rare. They are myoma, osteoma, enchondroma and cysts.

The tumors of the spinal cord which originate in the spinal canal outside the dura mater, but do not start from the vertebræ, are lipoma, which is congenital, fibroma, hydatid cysts, tuberculoma, neuroma and sarcoma.

In my list of forty-seven tumors involving the cord, about 10 per cent. were carcinoma or sarcoma and of vertebro-spinal character. About 12 per cent. were extra-dural but not of vertebral origin. The remainder were intra-dural tumors and were either of meningeal origin or were intra-medullary and of gliomatous character.

Symptoms and Course.—Vertebro-spinal tumors occur oftenest in later life, although sarcoma may occur early. Several of the vertebræ are usually involved. The bony tissue becomes softened and destroyed and a kyphosis occurs. This is not sharp and angular as in Pott's disease, but is more rounded. As the disease progresses it involves the nerve-roots and cord, causing local pain, rigidity of the spinal muscles, spasmodic twitchings, later anæsthesia, paraplegia and loss of control of the sphincters;

vasomotor and trophic disturbances also develop. In fine, the symptoms are like those of tumors of the spinal cord, to be described later, beginning with local pain and irritation and ending in a transverse or diffuse destruction of the nervous centres. The disease is very painful and often progresses rapidly, but remissions may occur.

Diagnosis.—The history of a primary malignant disease, the excessive pain, tenderness of the vertebra to jarring, then the rounded kyphosis, the progressive development of irritative and later of paralytic changes due to involvement of the cord make the diagnosis easy. The X-ray is often of decisive value. Examination of the cerebrospinal fluid is useful especially in excluding syphilis and inflammatory changes. The condition must be differentiated especially from tuberculosis of the spine and from spinal softening due to toxic conditions.

In the cases of sarcoma, operation is sometimes helpful. I have seen apparent cure by the use of Coley's toxins. The value of radium and of cross-firing with X-rays has yet to be established.

SPINAL CORD TUMORS PROPER

Extra-dural tumors of the spinal cord are about as frequent as intradural tumors. If, however, we leave out the vertebral tumors we find that neoplasms starting within the dura are at least five times as frequent as those starting from or involving its outer surface.

Of the tumors that start within the dura some originate from the meninges and some within the cord itself. Those originating from the meninges are rather more numerous. This statement would not be true, however, if we included under this head cases of syringomyelia. Including this disease, in my experience gliomatous tumor is the most frequent of all forms of those originating within the dura mater. This would correspond with Cushing's experience with brain tumors. He found that 65 per cent. of these were of gliomatous character.

Symptoms.—No disease of the spinal cord offers such a varying and often baffling group of symptoms as does that of spinal tumor; at least this is true of perhaps 10 per cent. of the cases. In the remainder of more or less typical cases the general symptoms are about as follows:

First, there is a group of symptoms extending from a certain fixed point in the spinal cord and rather steadily progressing for a period of months. Second, these symptoms are usually at first unilateral and then extend to the other side. Third, the symptoms are those of pressure and irritation with intense pain and signs of motor irritation, such as twitching and rigidity, followed later by symptoms of cord injury due to pressure and disintegration of the cord, causing paralysis, atrophy and loss of sphincter control. Fourth, there is evidence of total disintegra-

tion of the cord at a certain level, and associated with this are some evidences of bony disturbance, such as tenderness and rigidity and change in the percussion note. These general symptoms vary with the location, character, size and rate of growth of the tumor. Pain appears early and is a very frequent but not invariable symptom. It is severe and shooting and is of the root-pain character, being not distributed along different peripheral nerves but according to segments. There is usually a girdle sensation. In tumors of the cervical region this is often felt several segments below the level of the tumor. Numbness, hyperæsthesia and later anæsthesia below the level of the lesion occur. In intra-medullary tumors the symptoms may ascend. There is tenderness over the spine and rigidity at times. As the tumor increases, spastic conditions of the extremities develop, involving one or both legs or an arm and leg, later paraplegia, atrophy and loss of control of the bladder and rectum with bed-sores; then death ensues from exhaustion.

When the disease is cervical, first one, then the other upper extremity is involved, with evidence soon of implication of the pyramidal tracts. Then the legs become weak and stiff. The ciliary centre may be irritated or paralyzed if the tumor is in the lower cervical or first dorsal region. If lower down, there develops a hemiparaplegia, later a complete paraplegia, usually with exaggerated reflexes. If the tumor is in the lumbar region the reflexes are sooner lost and the sphincters early involved. In that site the tumor gradually grows and symptoms progress until the patient becomes paralyzed with anæsthesia, contractures, loss of sphincter control, bed-sores and finally death from intercurrent disease or exhaustion.

A rather frequent type of symptoms caused by spinal tumors, and occasionally by injuries and syphilis, is that known as a *Brown-Sequard paralysis* or hemiparaplegia. In a typical case of this kind there are paralysis of motion and muscle sense with exaggeration of deep reflexes on the side of the lesion, paralysis of cutaneous sensation, especially of pain and of temperature on the opposite side up to just below an anæsthetic band on the side of the lesion. On the side of the lesion the temperature may be slightly raised and there is often hyperæsthesia. There may be a band of anæsthesia at the level of the lesion and on the same side (Fig. 147). The symptoms may be grouped as follows:

On the side of the lesion:

1. Paralysis more or less complete.
2. Diminution of deep sensibility.
3. Tactile hyperæsthesia.
4. Vasomotor paralysis.
5. Sometimes a band of cutaneous anæsthesia just at the site of the lesion.

On the side opposite the lesion:

1. Diminution of cutaneous sensation, especially pain and thermic.

If the lesion, however, is low in the lumbar and sacral cord there is anæsthesia of both sides.

If the lesion is in the cervical region there will be a *spinal hemiplegia*, and perhaps pupillary changes.

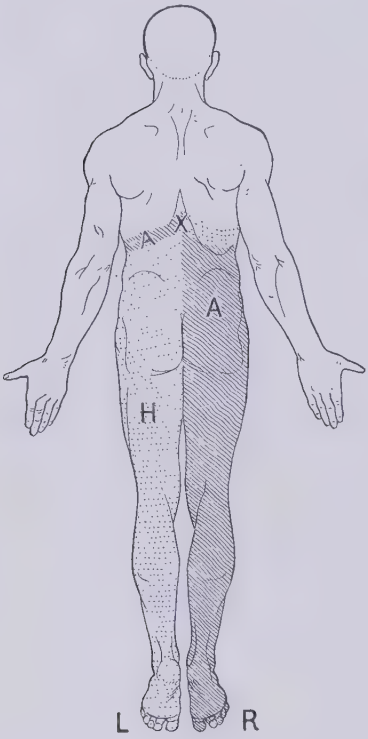


Fig. 147.—Showing the condition in a Brown-Séquard paralysis due to a tumor growing in the left side of the spinal cord. On the left side, hyperæsthesia, ataxia, paralysis, exaggerated reflexes. At the upper limit is a band of anæsthesia. On right side, anæsthesia.

The diagnosis of vertebro-spinal tumors from the extra- and intradural is not always possible. Root pains which begin unilaterally and later become bilateral indicate tumor of the spinal meninges. However, when extra-medullary growths lie in front of the cord there may be no pain, and if tumors involve the anterior roots there will be muscular atrophy of the root distribution.

In extra-medullary tumors there is apt to be more evidence of irritation such as increased amount of pain and reflex spasm of the lower limbs.

The Brown-Séquard syndrome may be due to a tumor either outside or inside the dura, but it is usually, at least, extra-medullary.

Experience has shown that there is a tendency to localize the spinal tumors below the level where they actually lie and it is absolutely important to make very careful tests as to the existence of hyperæsthesia, and anæsthesia in order to be sure of the tumor level. The existence of definite root pains correctly localized or of definite anæsthesia of the root type is characteristic of a tumor and distinguishes it from local meningitis.

The duration of life in cases of spinal tumor ranges from three to five years.

Pathological Anatomy.—This subject has been dealt with in the general introduction.

Spinal tumors are small in size, ranging from one-fifth to one and one-fifth inches (one-half to three centimeters) in diameter. The glioma may diffuse for a long distance through the centre of the cord, forming cavities (syringomyelia). The sarcomata may likewise be irregularly

spread along the surface of the cord. Spinal tumors are usually single, but fibromata, the parasites, cancer and sarcomata may be multiple.

Diagnosis.—The disease has to be distinguished from vertebral caries, localized serous cysts, transverse myelitis, syphilitic meningitis and pachymeningitis. The points to be noted as regards caries are the absence of an angular deformity or kyphosis; the small degree of pain, tenderness and rigidity, absence of anæsthesia, the age and the absence of tubercular diathesis. The slowly progressive course, beginning with pain, the one-sided motor and sensory paralysis and the localization of the symptoms exclude myelitis. Lumbar puncture may help in diagnosis by exclusion. The fluid shows no cells, but is often of a greenish-yellow tinge and has excess of globulin but no excess of cells.

Fig. 148.—Sarcoma of cord and cervical region.

The character of the tumor cannot often be determined certainly. The probabilities are in favor of sarcoma or glioma, especially in middle life. Syphiloma may be determined from the history and results of lumbar puncture. Tuberculoma is very rare, and is more likely to occur in the young.

Prognosis.—This is not good. Medicine is of little use except in syphiloma, and even here it may be ineffective. Tubercle may perhaps cease to grow. Surgical interference now saves the life of some patients and is a more hopeful form of interference on the whole than in cerebral tumors. This is more true now than ten years ago. Out of eight rather recently operated cases, there were excellent results in five. These were, to be sure, selected cases of intradural or non-vertebral tumors.

Treatment.—In syphilitic tumors appropriate remedies may do good and should be vigorously used. In tubercle, bodily rest, tonic treatment, iodine and cod-liver oil may be of service. In gliomata and sarcomata, arsenic may be tried. In other forms, symptomatic treatment is all that can be recommended medically. In all cases of spinal tumor surgical interference should be considered; extra-dural and extra-medullary tumors

can often be removed with success and also with benefit to the patient. In medullary tumors operations have been successfully done by the process of incising the cord and allowing the tumor to extrude (decompression method of Elsberg).

The use of radium and cross-firing with X-ray may prove to be helpful.

CAVITIES IN THE SPINAL CORD

The cavities of the spinal cord are known as:

1. Hydromyelia.
2. Myelitic cavities.
3. Syringomyelia.

There may be various combinations of those processes, the most common being a combination of hydromyelia and syringomyelia.

HYDROMYELIA

This is a dilatation of the central canal of the cord, the cavity being filled with fluid (Fig. 149). The condition may be associated with

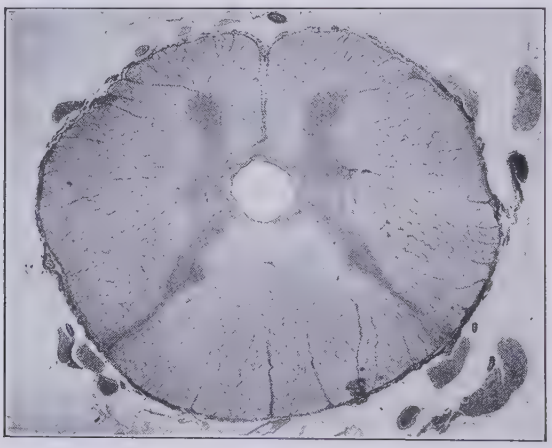


Fig. 149.—Hydromyelia.

hydrocephalus or with spina bifida, or it may be independent of these conditions. The dilatation may be cystic and irregular, or, as is more usual, may extend throughout the cord. The dilatation extends more posteriorly, because the posterior columns are formed latest. The abnormality may be slight and give rise to no symptoms, or a pathological process like a gliomatosis may develop upon it.

MYELITIC AND HEMORRHAGIC CAVITIES

Such cavities may be formed in the cord by a central excavating myelitis or by small hemorrhages. These cavities are usually small,

irregularly distributed, and are lined with connective tissue. They may be formed in rare cases in connection with hydromyelia or neoplasms.

GLIOSIS AND SYRINGOMYELIA

Syringomyelia is a disease of the spinal cord characterized by a development of gliomatous tissue in the central parts with formation of cavities. Such cavities are not always formed, however, and then the condition is known as spinal gliosis. Clinically the condition is characterized by the slow, progressive development of a muscular atrophy with peculiar disturbances of sensation and of vasomotor function.

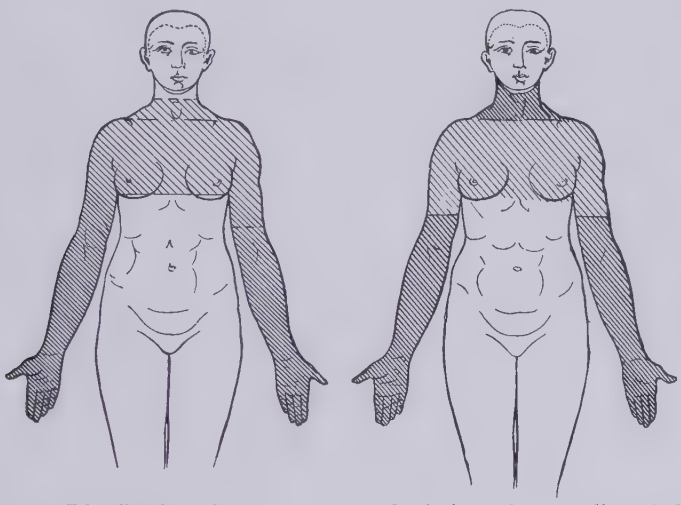


FIG. 150.—Distribution of cutaneous anæsthesia in syringomyelia. 1 shows area of analgesia, 2 shows that of thermo-anæsthesia. The darker shades show where there is anæsthesia to pain and temperature, the tactile sense being unimpaired.

Etiology.—The disease is rare. It is more frequent than amyotrophic lateral sclerosis and less frequent than multiple sclerosis. It occurs oftener in men than in women, and develops in early and middle life, between the ages of fifteen and forty-five. It occurs especially in persons who follow hard manual occupations, such as butchers, tailors, etc. Traumatism is the most important exciting factor in my experience. There is sometimes an underlying teratological defect in the cord. Infectious diseases seem occasionally to start up the trouble. Heredity, syphilis and alcohol are not causal factors.

Symptoms.—The disease begins insidiously with some aching pains and paræsthesia. If it starts in the cervical cord, there is soon a muscular atrophy of the hands resembling spinal progressive muscular atrophy, but with anæsthesia. As the disease progresses the weakness

and atrophy of the hand muscles become more noticeable and gradually extend toward the trunk. The atrophy often comes on in both extremities at about the same time. Fibrillary contractions and partial degeneration reaction may be observed. Cutaneous anæsthesia of the affected hand and arm to temperature and pain, but not to touch, takes place; and this is so marked as to be almost pathognomonic of the disease.

Fig. 151.—Curvature of spine in syringomyelia. (*Erb.*)

The legs do not become affected until late, and then generally show a spastic paraplegia. The throat and face are rarely involved. There is a scoliosis of the spine, generally in the dorso-lumbar region (Fig. 151).

Vasomotor, secretory, and trophic symptoms are prominent. The hands may be œdematous or red and congested. Sweating or dryness of the skin may occur. Eruptions appear on the skin, such as bullæ, herpes and eczema. Painless whitlows attack the fingers and may destroy the terminal phalanges. Erosions and ulcerations also occur. The nails become dry, brittle and drop off. Arthropathies and spontaneous fractures have been observed. The pupils may be unequal and the bulb retracted.

Late in the disease symptoms of involvement of the medulla develop. At this time also the bladder, rectum and genital centres are attacked. The disease progresses slowly for years, with remissions of various degree.

The cardinal symptoms are a progressive muscular atrophy, with a peculiar disassociated cutaneous anæsthesia, trophic disturbances, and scoliosis.

Pathological Anatomy.—The seat of the disease is the substance of the cord. On opening it by transverse sections one finds one or more cavities extending in various degrees up and down. The most frequent primary site is the cervical swelling. From here it usually passes down and may reach the whole length of the cord. It also extends upward and

may involve the medulla and the nuclei of the cranial nerves. The cavities are of irregular shape, small size and filled with a liquid like the cerebrospinal fluid. They are situated oftenest posterior to the commissure and involve one or both posterior horns, but they may be so extensive as to involve almost the whole of the centre of the cord at some levels. The walls are usually lined by a membrane and surrounded by a gliomatous tissue (Figs. 153–156).

The *glia* cells are in various stages of development and degeneration. In parts of the cord the new growth may form a large and solid mass occupying most of the centre of the cord. Small hemorrhages and foci of myelitis may be present. In some cases there is evidence of a dilated central canal, with neuroglia hyperplasia of the walls and a gliomatous

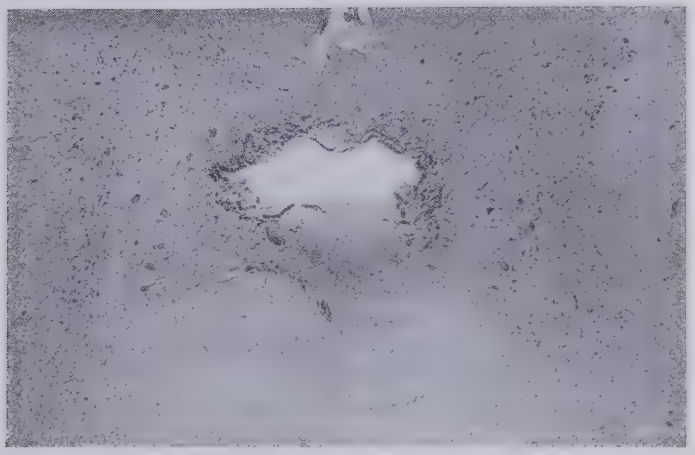


Fig. 152.—Central canal of cord with beginning syringomyelia.

infiltration about this. The epithelium of the central canal may form part of the wall of the cavity.

Gliomatous tissue is composed of small cells, round or of irregular shape, with a large nucleus and fine fibrillary prolongations. In glioma these cells are relatively much more numerous, while the fibrillary network is less conspicuous. When the fibrillary substance is more prominent we have gliosis, and when it is almost exclusively fibrillary we have the condition found in multiple sclerosis. In glioma and gliosis the tissue is penetrated by small blood-vessels whose walls are often diseased, so that minute hemorrhages occur and the glioma becomes stained and pigmented. In other cases it is gray or yellowish in color.

The rich cellular proliferation in gliomata has suggested an analogy in its growth to that of inflammation, and the term gliosis is used as analogous for neuroglia to sclerosis of connective tissue. In gliosis there

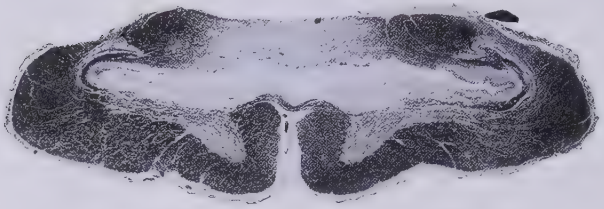


FIG. 153.—Syringomyelia, dorsal segment.

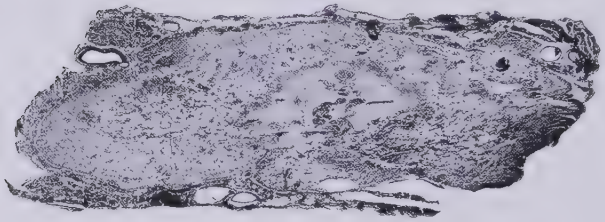


FIG. 154.—Total syringomyelic degeneration, dorsal segment.

FIG. 155.—Syringomyelia, dorsal segment.

FIG. 156.—Syringomyelia, lumbar segment. (*From microphotographs by Dr. C. I. Lambert.*)

is a tendency to softening and formation of cavities, and all nerve-fibres disappear. In multiple sclerosis some nerve-fibres remain, and one observes the presence of granular and amyloid bodies.

Peculiar Types.—1. The disease may be latent, giving rise to very few symptoms or to none that are characteristic.

2. There may be a period of irritation and pain in the extremities followed by paraplegia, with few sensory troubles, the course suggesting a chronic transverse myelitis or a Brown-Séquard paralysis.

3. There is a type in which bulbar symptoms develop early, but differing from ordinary bulbar paralysis in the involvement of the trigeminus and other cranial nerves not commonly attacked.

4. There is a type characterized by the symptoms of muscular atrophy with analgesia and felons (Morvan's disease).

Diagnosis.—The disease is distinguished in its classical form by (1) its very slowly progressive course with periods of latency and remission; (2) by the progressive muscular atrophy combined with the peculiar dissociated disturbances of sensibility; (3) by the trophic disturbances and scoliosis.

It has to be distinguished from progressive muscular atrophy and dystrophy, from amyotrophic lateral sclerosis, hypertrophic cervical pachymeningitis, chronic transverse myelitis, Morvan's disease and anæsthetic leprosy.

The sensory and trophic disorders and scoliosis enable one to distinguish it from progressive muscular atrophy. In leprosy the dissociation of the sensory symptoms is not present, and the anæsthesia is distributed along the course of the nerves or in sharply circumscribed plaques. In some cases the peculiar tubercular disease of the skin and the history of the case make the diagnosis easy. In leprosy, also, there is a perineuritis, and the enlarged inflamed nerves may be felt. Portions of the skin may be excised and examined for the leprosy bacillus. As regards the differentiation from Morvan's disease, this cannot often be done. Still whitlows are rare in ordinary forms of syringomyelia. Morvan's disease begins in one hand and slowly extends, with remissions, to the other. Usually there is loss of tactile as well as thermic and pain sense.

The prognosis, so far as cure is concerned, is bad; but the disease has often a long course, ranging from five to twenty years, and periods occur in which the progress of the disease seems arrested and improvement occurs.

Treatment.—It is not impossible that we may find some drug which acts specifically on gliomatous tissue, checking its growth. At present we know of only two things which may possibly do this: nitrate of silver and arsenic. These drugs should be given; and for the rest, tonic and

symptomatic treatment is indicated. Intra-spinal medication by lumbar puncture may eventually furnish us help in controlling the disease. The use of radium and of cross-firing with X-ray is being used with some apparently good result. The exposure of the spinal cord and decompression by the method of Elsberg has been suggested for gliosis. It has been successful in isolated gliomatous masses. Early diagnosis and early attention to treatment are most important.

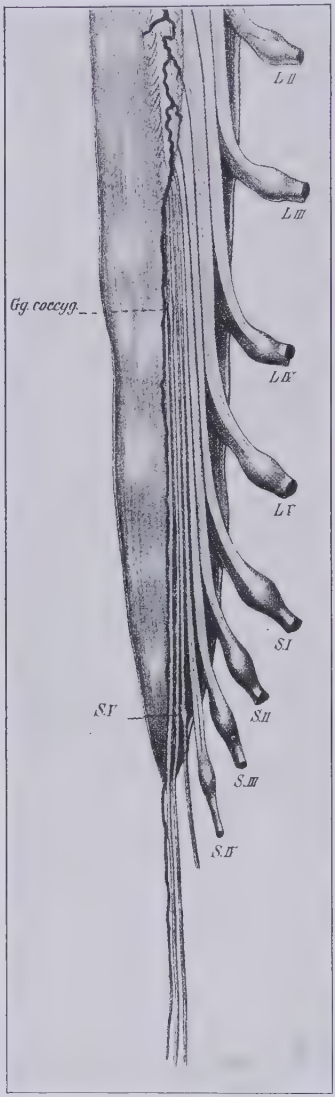


FIG. 157.—Showing left-half of cauda equina, and tip of spinal cord just at level of 3d-lumbar root.

THE RECOGNITION OF DISEASES OF THE CAUDA EQUINA

Anatomy.—The cauda equina is made up of the five lumbar, five sacral and one coccygeal nerve-roots. They lie within the dura mater forming a thick bundle and extending down the vertebral canal for 14 cm. They are still distinct motor and sensory roots, and do not unite till they have passed out of the dura. The cauda begins at the lower tip of the cord, at the level of the lower edge of the second lumbar vertebra. The term conus is applied to the lower end of the cord and includes the parts below the second, or some say third, sacral segment. The cord here becomes much smaller, loses some of its distinctive microscopical structure, and the anterior root-fibres are so much smaller and less numerous than the posterior or sensory that their destruction causes no marked paralysis.

The arrangement of the segments of the cone and the nerves is shown in Figs. 157 and 158. The arrangement of the visceral centres is given by Müller as follows: second sacral, erection centre; third sacral, ejaculation centre; fourth sacral, bladder (detrusor) centre; fifth sacral, sphincter ani centre. The distribution of the sensory nerves is shown in the Figs. 127 and 128.

Cauda lesions are generally due to tumor, pressing on the part, neighboring bone disease, and injuries. A caudal root neuritis has been described (Elsberg, Kennedy), simulating cauda tumor.

The symptoms are those of pressure with irritation, and later destruction of the cauda nerve-roots. The symptoms usually come on slowly (tumor and bone disease) with severe root pains, mostly in the sacral nerve distribution, sometimes in the bladder and rectum. They

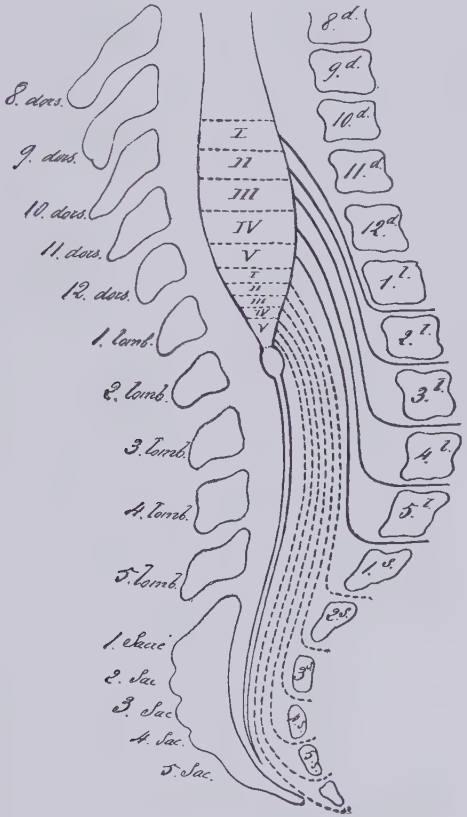


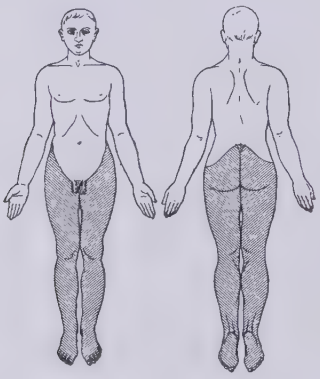
Fig. 158.—Showing arrangement of segments and nerves of cauda equina.

are bilateral but more on one side. Later and less notable are motor symptoms: weakness of the legs and loss of ankle jerks. The sensory symptoms remain in the foreground. There is hyperæsthesia, then anæsthesia not symmetrical on the two sides and not sharply segmental as when the cord is involved.

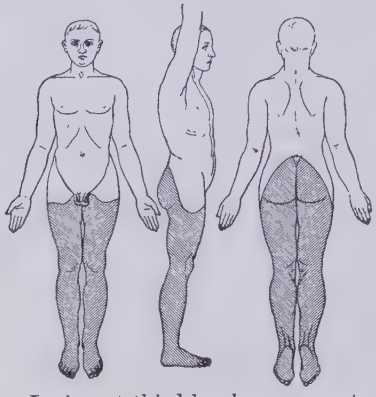
Paralysis of the legs and of the bladder, sexual and rectal centres comes on eventually but usually the conus is then also involved.

Cauda lesions must be differentiated from cord and especially conus

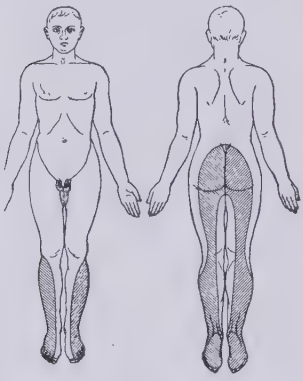
lesions (second to fifth sacral segments). In conus lesions we oftener have an acute onset from myelitic softening or hemorrhage, and in this case there is little pain. Fibrillary contractions and muscular twitchings occur. If the lesion does not reach *above the conus* there is little paralysis. Symmetrical anæsthesias develop in areas corresponding to the height of the lesion (Fig. 159), and there is sometimes a differentiation of sensibility,



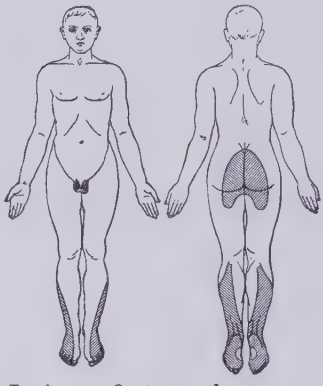
Lesion at second lumbar segment.



Lesion at third lumbar segment.



Lesion at fifth lumbar segment.



Lesion at first sacral segment.

FIG. 159.—Lesions at different levels of the lumbar and sacral cord, showing areas of anæsthesia. (*Müller.*)

which does not occur in cauda lesions. The bladder, sexual and rectal centres are early involved.

In lesions of the peripheral nerves, the trouble (usually neuritis or injury) comes on rather rapidly. There are sciatic pains, tender points, the lesion may be only unilateral, the pains are not so severe. There is little or no paralysis and no involvement of the visceral centres; the sensory and motor symptoms go together, the sensory slightly predominating; there is often a history of sciatica and of alcoholism, or injury.

CHAPTER XVI

ANATOMY AND PHYSIOLOGY OF THE BRAIN

Historical.—The first artistic and approximately correct illustrations of the brain were made in the middle of the sixteenth century (Fig. 160.) About 100 years later, the details became more accurate (Fig. 161) and in the next century, the eighteenth (Fig. 162), there is a modern fineness of finish without yet absolute accuracy, which was only achieved in the nineteenth century.

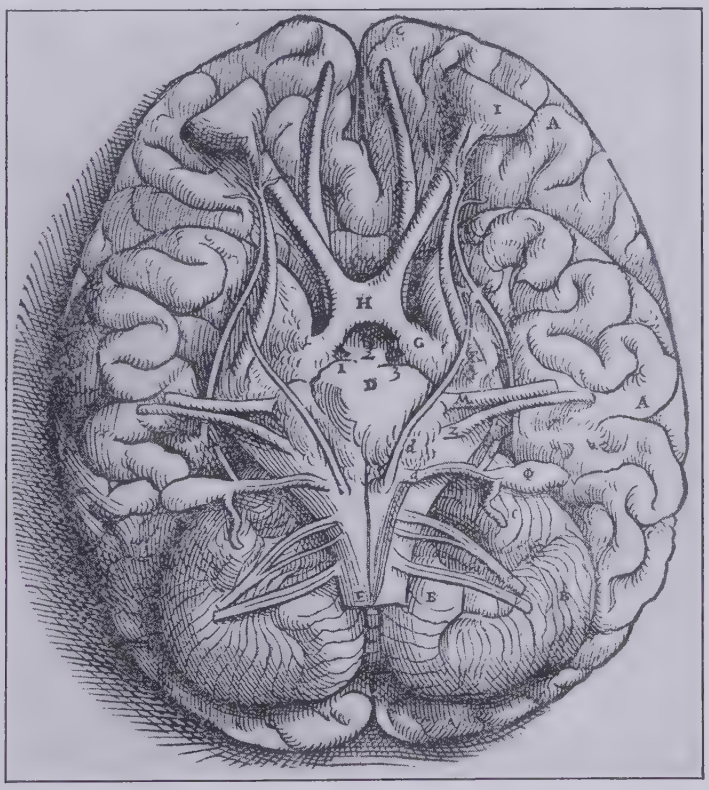


FIG. 160.—The brain. (*Vesalius*, A. D. 1555.)

Anatomy.—The nervous system is developed from a hollow tube formed by a folding of the epiblast. The brain or encephalon grows out from its anterior part. This swells into three cavities called the anterior, middle and posterior cerebral vesicles. From the anterior a secondary vesicle develops; the posterior divides into two; so that eventually there are five vesicles. Out of them the different parts of the brain are formed.

1. From the anterior vesicle there grow the cerebral hemispheres, the corpus callosum and anterior commissure, fornix, corpus striatum and olfactory lobes. It includes also the anterior part of the region lying under the thalamus in which are the optic chiasm and pituitary body. These structures form the end-brain or telencephalon.

2. From the posterior part of the primary vesicle come the thalamus, pineal gland (or epithalamus), geniculate bodies (or metathalami), and some structures lying under the thalamus, viz., the corpora mammillaria and Luys' body. These parts form the 'tween-brain or diencephalon.

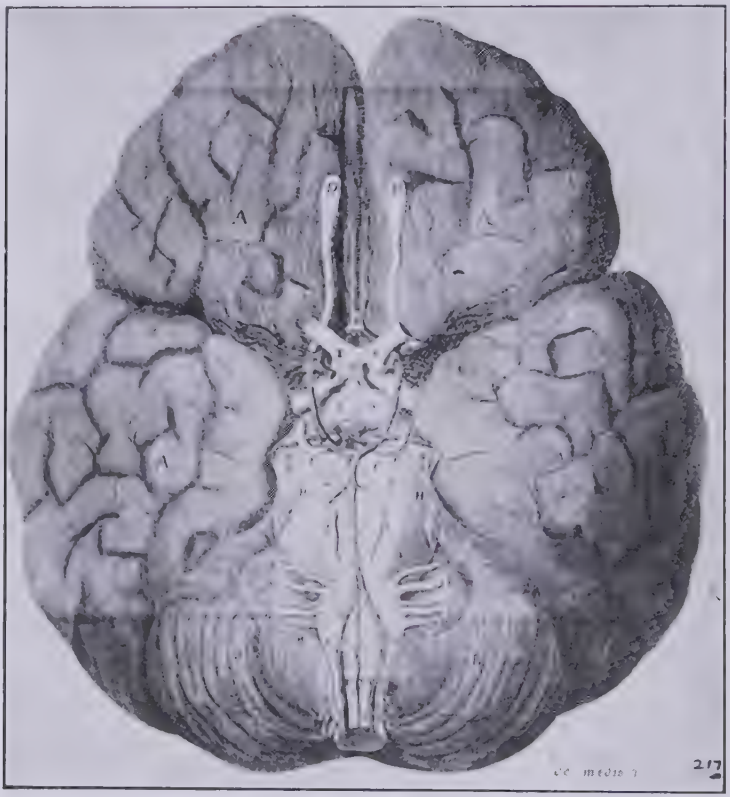


FIG. 161.—The brain, showing the circle of Willis. (*Willis*, circ. 1665.)

3. From the middle cerebral vesicle there grow the corpora quadrigemina and cerebral peduncles, with the red nuclei and substantia nigra. They form the mid-brain or mesencephalon.

4. From the fourth vesicle, which is a secondary vesicle developed from the third primary, come the "isthmus" (which includes the superior cerebellar peduncles and valve of Vieussens) and the "hind-brain," composed of the cerebellum, its middle peduncles and the pons. This part is also called the metencephalon.

5. The fifth vesicle (also a development from the third) forms the medulla oblongata, or after-brain, or myelencephalon.

The development of these parts is shown in the accompanying Figs 163, 164.

The arrangement in tabular form taken from Villiger's anatomy shows a slightly different arrangement as to the hind-brain.

In man the fore-brain is enormously developed, the 'tween-brain moderately developed, the olfactory lobes are atrophic, the mid-brain is almost rudimentary, the hind-brain well developed, the after-brain relatively not much developed. The divisions above given in detail seem somewhat academic and impractical as applied to adult human brains, but the main features are not so; indeed they are quite essential to the understanding of modern anatomy.

FIG. 162.—The brain. (*Ruysch*, A. D. 1724.)

End-brain or telencephalon.	Cerebral hemispheres, rhinencephalon and corpora striata.
'Tween-brain or diencephalon.	Optic thalamus and geniculate bodies.
Mid-brain or mesencephalon.	Cerebral peduncles and corpora quadrigemina.
Isthmus and hind-brain or metencephalon.	Cerebellum and its peduncles. Pons.
After-brain or myelencephalon.	Medulla.

Encephalon—Brain	**Prosencephalon**—Fore-brain.	Telencephalon. End-brain.	Hemisphærium. — Pallium. Rhinencephalon. Stem of End-brain.
			Pars optica Hypothalami.
		Diencephalon. 'Tween-brain.	Pars mamillaris Hypothalami.
			Thalamencephalon. — Thalamus. Metathalamus. Epithalamus.
	Mesencephalon—Mid-brain.	. . . . .	Pedunculi cerebri. Corpora quadrigemina.
	Rhombencephalon—Hind-brain.	Isthmus rhombencephali.	
		Metencephalon or hind-brain.	Cerebellum. Pons.
		Myelencephalon after-brain.	Medulla oblongata.

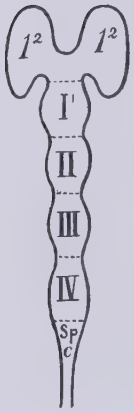


Fig. 163.

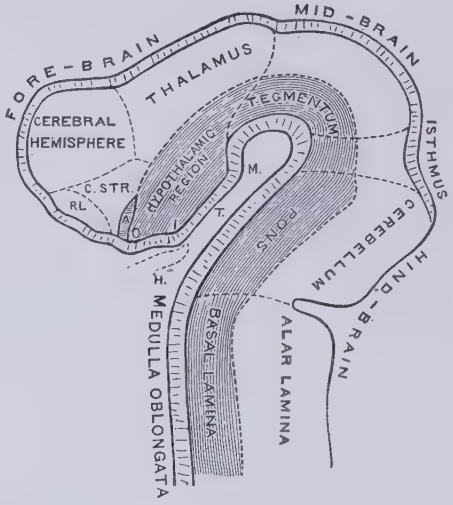


Fig. 164.

Fig. 163.—The cerebral vesicles.

Fig. 164.—Median section through the brain of a human embryo at the end of the first month. (*His.*)

In the process of development of the brain, the neural canal becomes variously enlarged and constricted, until the ventricles of the brain, the foramina of Monroe, and the aqueduct of Sylvius are formed. The ventricles are the two lateral, the third, fourth and fifth. The foramina of Monroe connect the lateral and third ventricles; the aqueduct of Sylvius connects the third and fourth ventricles.

Along the mesial or inner surface of each optic thalamus runs a groove known as the *fissure of Monroe.* This, according to Minot, may be traced along the sides of the aqueduct of Sylvius and fourth ventricle into the spinal cord, where it is identical with the line of division that in embryonic life separates the dorsal from the ventral parts of the cord, forming the dorsal and ventral zones of His. Minot regards this line of demarcation as having great morphological importance. All parts of the brain and cord dorsal to it form a dorsal zone and include the receptive part of the spinal

cord and medulla, as well as of the cerebellum and cerebral hemispheres. This dorsal zone contains only nerve-cells whose processes never leave the nerve-centres to form nerves, and it receives all the entering sensory nerve-fibres. It is the recipient part of the nerve-centre.

The ventral zone contains all cells whose processes go out to form peripheral nerves; it has also some cells of the other type, but it does not receive any entering sensory nerve-fibres. It is the efferent part of the nerve system and it includes the anterior and part of the lateral columns of the spinal cord, as well as the ventral parts of the mid-, hind-, and after-brains.[1]

The different segments of the brain are composed of deposits of nerve cells forming gray matter or ganglia, and of strands of nerve-fibres connecting these ganglia. Most of the general description of these parts must be gotten from treatises on anatomy.

The Cortex Cerebri and the Convolutions.—The gray matter of the surface of the brain is called the cortex cerebri, and it is by far the largest and most important deposit of nerve-cells in the body. The cortex is from 2 to 4 mm. (one-twelfth to one-fifth of an inch) thick, and its total superficial area is 1,800 to 2,700 sq. cm. The area of gray matter lying in the fissures is about twice that lying on the surface (Donaldson). The cerebral cortex is arranged in folds or convolutions (gyri) separated by fissures or sulci. These fissures divide the brain also into lobes. The fissures of the brain are divided into *primary* and *secondary*. The former are permanent, and present little change in size, location or direction. The latter are variable in all these respects, and are often called sulci for purposes of distinction.

The primary fissures of the brain are:

The longitudinal.
The transverse or choroidal.
The fissure of Rolando or central.
The fissure of Sylvius.
The parietal.
The parieto-occipital.
The calcarine.

The position of these fissures is indicated in the accompanying diagrams, which are based on descriptions of Eberstaller (Figs. 167, 168, 169).

The primary fissures divide the cerebrum into various portions called lobes. The lobes are:

Frontal.
Parietal.
Temporal.
Occipital.
The central or island of Reil.
Olfactory.
Limbic.

There are eight fissures and seven lobes. The position and arrangement of these are indicated in the illustrations.

Microscopical Anatomy of the Convolutions.—The cortex of the cerebrum is composed of nerve-cells, a network of nerve-fibres and processes, and of neuroglia tissue. Superimposed upon it is a very vascular membrane, the pia mater, which sends a rich plexus of vessels into it.

The Fibre Layers of the Cortex.—The nerve-fibres of the cortex are arranged in more or less definite zones or layers. These layers are very well marked in some parts of the cortex, less so in others. They have received different names, but adopting the nomenclature and classification of Campbell, the principal zones are:

[1] Dr. Adolph Meyer has described a segmental division of the nervous system, based on function. Johnson has devised another scheme of longitudinal division in accordance with function. These are efforts in a right direction but not yet successful.

1. An outermost fibreless or neurolgia layer.
2. Zonal or tangential layer.
3. Line of Baillarger; in the occipital cortex called the line of Glenari.
4. Radiary zone.

Passing into the cortex at right angles to these layers are the radiations of Meynert; and below the cell layers are the association fibres of Meynert.

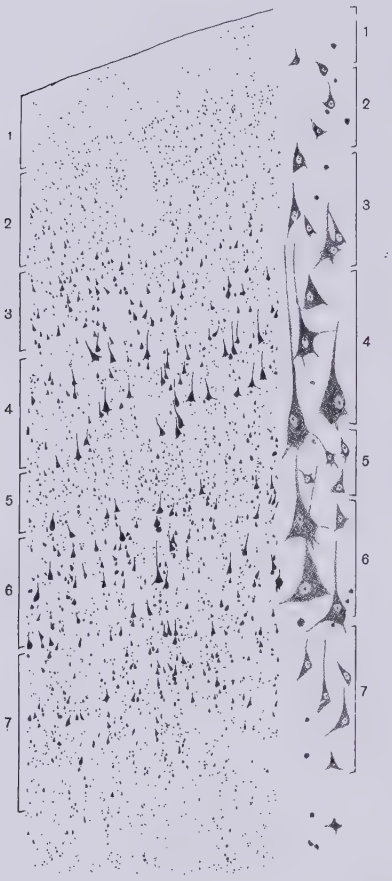


FIG. 165.—Type of cell lamination in the post-central area. (*Campbell.*)

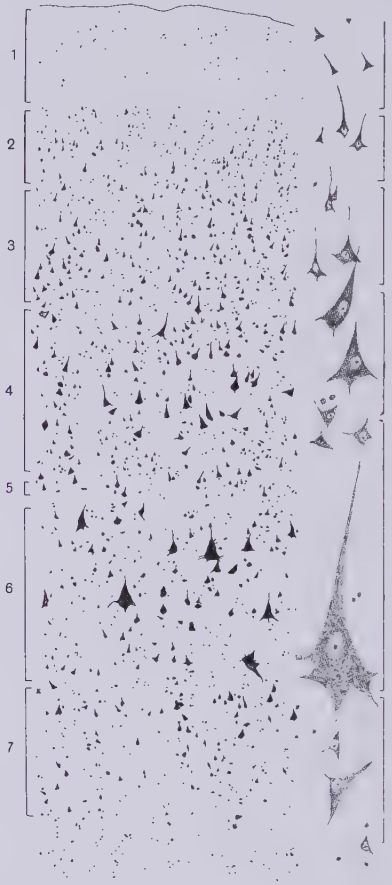


FIG. 166.—Type of cell lamination in the precentral area (*Campbell*). In the sixth layer is seen a cell of Betz.

Cell Layers.—The cells of the cortex are also arranged in layers more or less definitely marked, and varying in different parts of the cortex. The type of arrangement as given by Campbell is as follows:

1. The plexiform layer containing the cells of Cajal. It is also called the molecular or first layer.

2. The layer of small pyramidal cells; the cells here are closely packed, and while most of the cells are pyramidal, some are polymorphous. This is called also the second

layer. The cells are about the size of a white blood-cell; this layer is fused with the third layer.

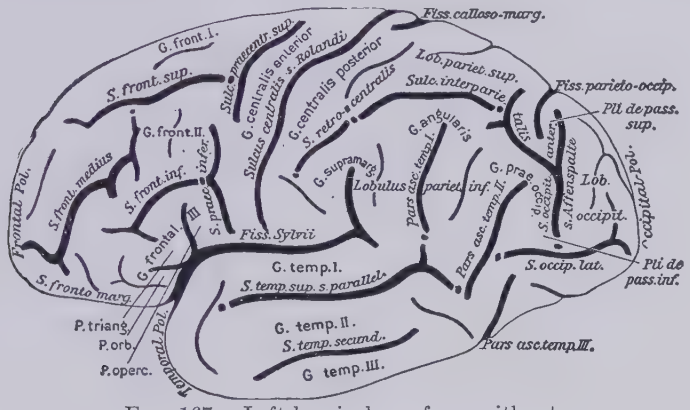


FIG. 167.—Left hemisphere, from without.

3. Layer of medium-sized pyramidal cells. Here the cells are all pyramidal and increase in size from above downward.

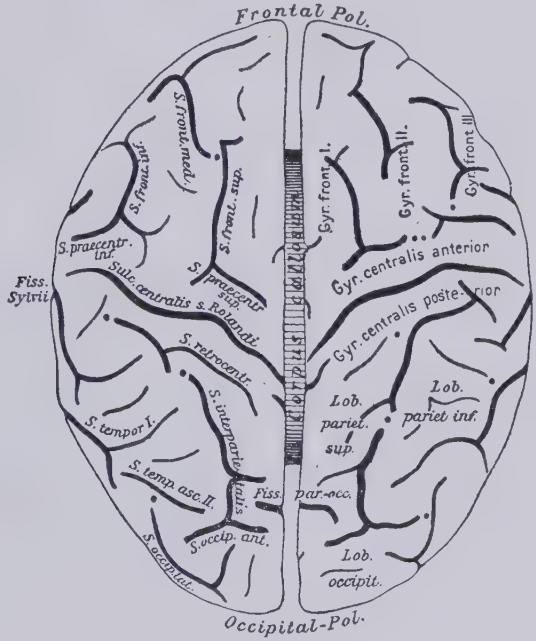


FIG. 168.—The convolutions from above.

4. Layer of large pyramidal cells, lying on a level with the line of Baillarger.

5. Layer of stellate cells, known also as the granule layer or Körnerschicht. This is best represented in the calcarine cortex.

6. Internal layer of large pyramidal cells; this contains the giant cells of Betz, and in the visual cortex the large solitary cells.

7. Layer of spindle cells.

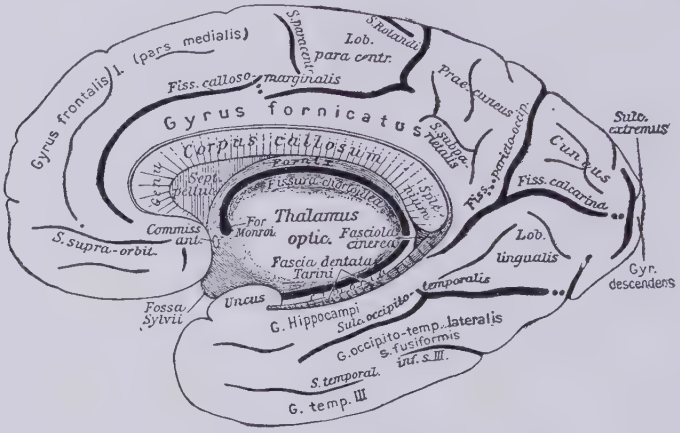
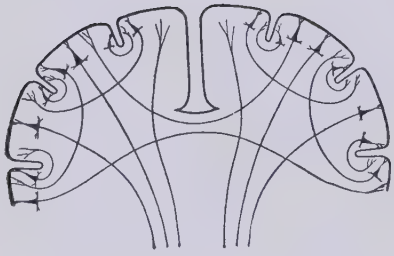
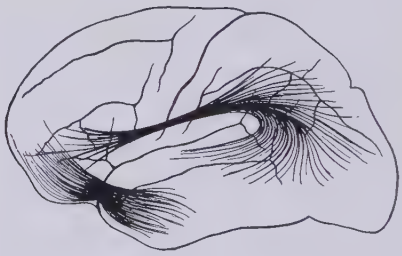


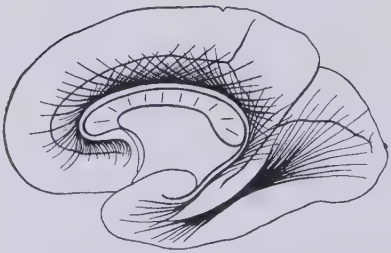
FIG. 169.—Mesial surface.



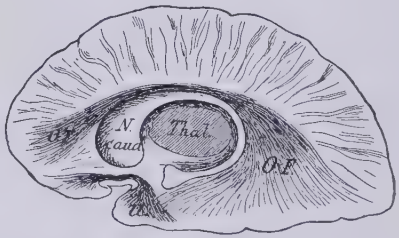
Association, commisural and projection fibres.



Uncinate fasciculus below; superior longitudinal fasciculus above.



Cingulum above; inferior longitudinal fasciculus below.



Fasciculus occipito-frontalis superior

FIG. 170.—Association tracts of the brain. (*Villiger.*)

The small pyramidal and spindle cells measure about 10 × 18μ; the large, 20 × 40μ($\frac{1}{1200} \times \frac{1}{600}$ in.). There are in the precentral convolutions giant cells (of Betz) which measure 125 × 55μ($\frac{1}{200} \times \frac{1}{500}$ in.).

The cortical gray matter, as will thus be seen, contains layers of nerve-cells, into which nerve-fibres penetrate. These terminate, as do all fibres, in end brushes, which surround the receptive cells. An enormous number of fine fibres is given off

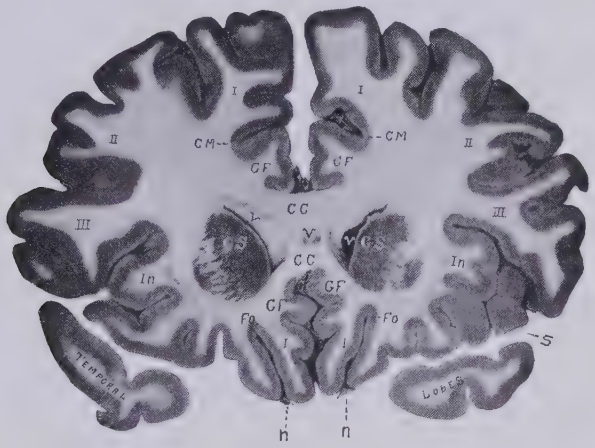


FIG. 171.—Vertical section through brain immediately behind the concavity of the corpus callosum. *I*, *II*, *III*, Frontal convolutions; *CC*, corpus callosum; *V*, lateral ventricles; *In*, island of Reil; *S*, fissure of Sylvius; *CS*, corpus striatum.

by the cells; some of these form layers in the cortex and connect neighboring parts, others run out and connect more distant parts or pass down to lower levels. There are thus three kinds of fibres—afferent, associative and efferent—just as there are

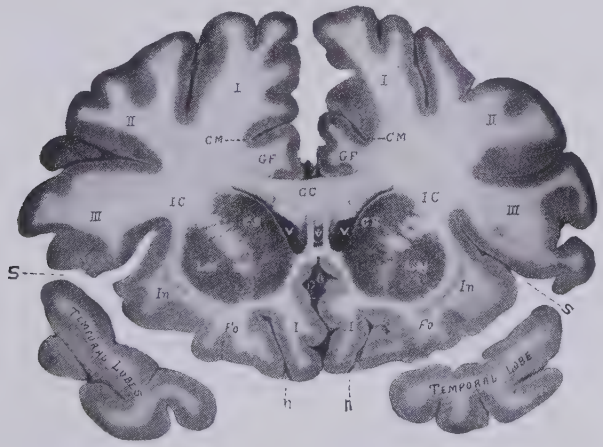


FIG. 172.—Section further back through caudate and lenticular nuclei, showing *IC*, internal capsule.

three types of cells, and since nerve-cells and fibres are really parts of the same unit—the neuron—there are practically three kinds of neurons in the cortex.

Association Fibres.—The different convolutions and lobes of the brain are connected with each other by association fibres (Fig. 170) and commissural fibres, and

FIG. 173.—Section just in front of anterior commissure.

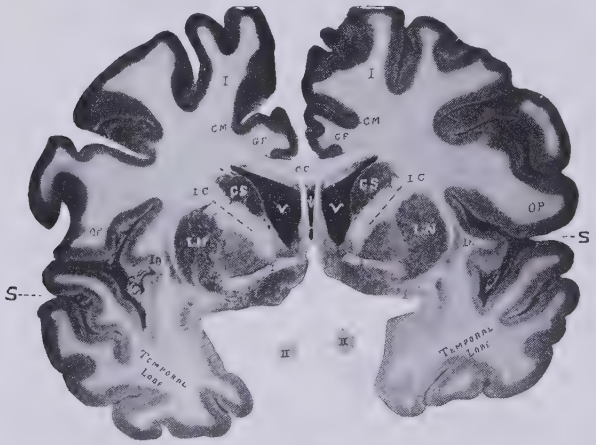


FIG. 174.—Showing divisions of lenticular nucleus. *OP*, Operculum; *II*, optic nerves.

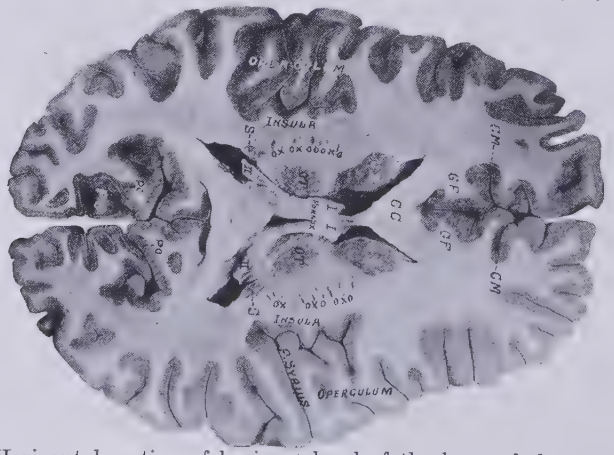


FIG. 175.—Horizontal section of brain at level of the knee of the corpus callosum, showing fornix.

to the ganglionic masses below them by projection fibres. The association fibres consist of short fibres connecting neighboring convolutions and of five long tracts which connect neighboring or distant lobes (Fig. 167).

The Central Ganglia of the Brain.—The relations of the cortex and of the ganglionic masses of the brain and of the different associating tracts is shown in a series of photographs made by Prof. J. C. Dalton over twenty years ago. Nothing so good has been done since his work.

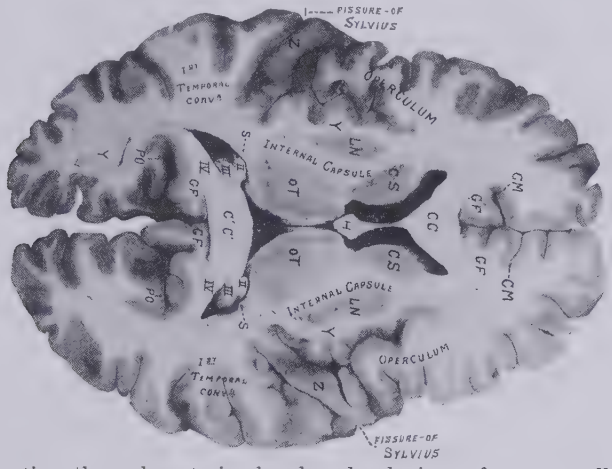


FIG. 176.—Section through anterior bend and splenium of corpus callosum. The fornix is nearly removed showing 3d-ventricle.

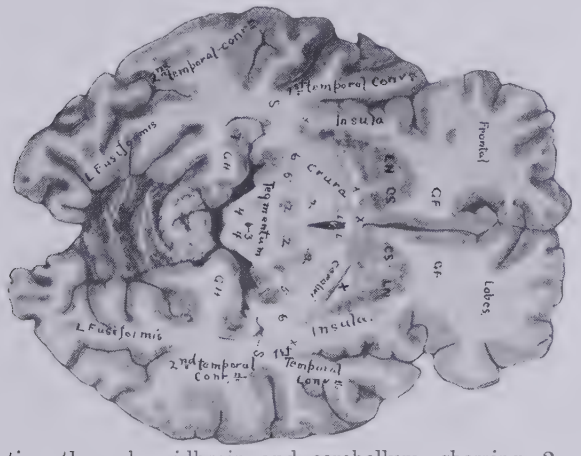


FIG. 177.—Section through midbrain and cerebellum, showing, 2, red nuclei, 3, acqueduct of Sylvius, 4, pons.

THE CEREBELLUM

In man it consists of a median portion or vermis and two lateral hemispheres. It is connected to the cord and rest of the brain by anterior, middle, and posterior peduncles. It is composed of an external layer or cortex of gray matter, of central white matter, and central ganglia. The gray matter lies in very close, nar-

row folds, producing with the white matter an appearance on section called the *arbor vitæ*. The vermis and hemispheres are divided by sulci into a number of lobes or lobules. The vermis is divided into superior and inferior portions. Its further subdivisions and those of the hemispheres are shown in the cuts (Figs. 178 and

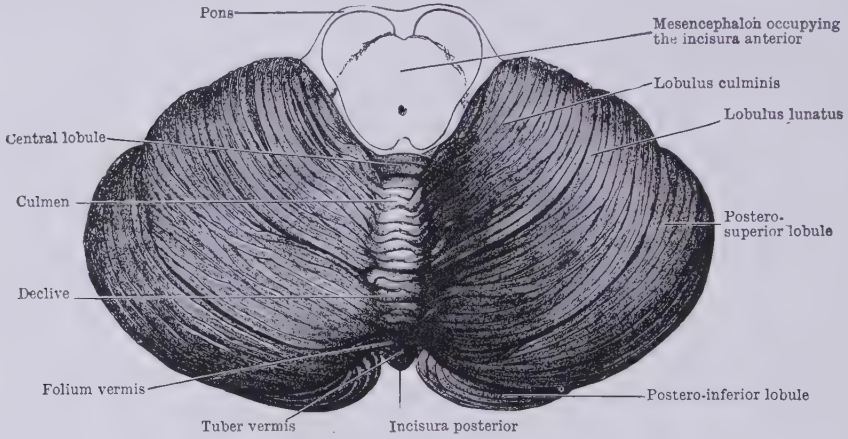


FIG. 178.—Upper surface of cerebellum. (*Cunningham.*)

179). In the white matter of both hemispheres is a nucleus of small multipolar cells, the corpus dentatus or ciliary body. To the median side of this, and belonging structurally to it, is a small nucleus, the emboliform nucleus. In the inferior vermis is a collection of larger multipolar cells, the *nucleus fastigium* or tegmental nucleus;

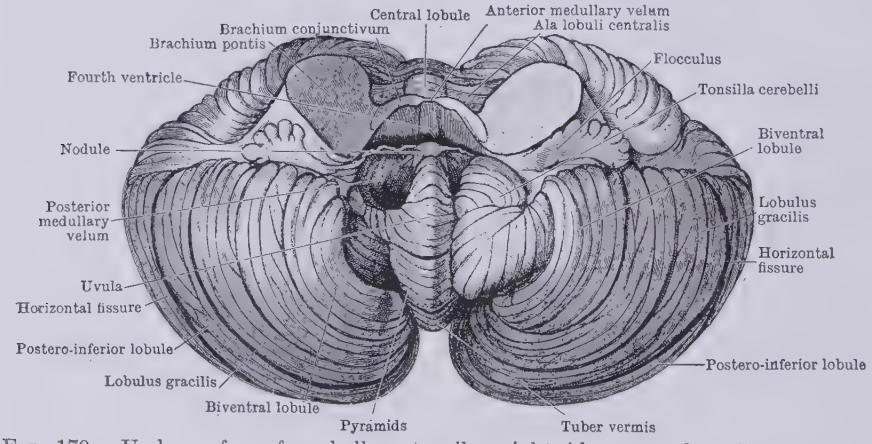


FIG. 179.—Under surface of cerebellum, tonsil on right side removed. (*Cunningham.*)

just to the outer side, between it and the emboliform nucleus, is a small collection of cells, resembling those of the *n. fastigii*, called the nucleus globosus.

The general arrangement of the cerebellar cortex is analogous to that of the cerebrum. Associative and receptive cells are found in the granular and molecular layers, and they send processes forming a rich network around Purkinje's cells, which are

efferent in function. The comparatively small number of the large cells is in harmony with the view that the cerebellum is an organ that receives and adjusts nerve impulses for co-ordinate distribution. All parts of the cerebellar cortex are anatomically alike.

The white matter of the cerebellum consists of nerve-fibres, some of which go to form the peduncles. Others form anterior and posterior commissures, running through the two extremities of the vermis and connecting the hemispheres. There is also a longitudinal commissure in the vermis. The white matter around the corpus dentatum is called the *fleece*.

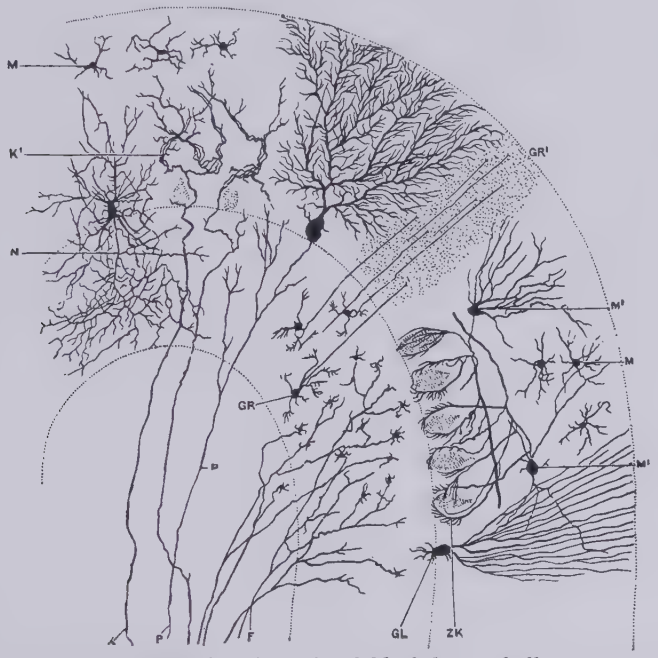


FIG. 180.—Transverse section through a fold of the cerebellar cortex. *P*, axon of Purkinje cell; *F*, moss fibres; *KK'*, fibres from white matter of cerebellar fold ending in connection with dendrites of Purkinje cells; *M*, small cells of molecular layer; *GR*, granule cells; *M'*, basket cells; *GL*, neuroglia cell. (*Cunningham.*)

THE GLANDS OF THE BRAIN

The pituitary body or hypophysis cerebri is a small oval body lying in the sella turcica or hypophyseal fossa. It is composed of an anterior and a smaller posterior lobe. It is connected to the tuber cinereum above by a stalk or infundibulum which extends downward and is attached to the posterior lobe. The anterior and posterior lobes are separated by a cleft; and a narrow strip of the posterior lobe bordering the cleft, has a special structure which is called the *pars intermedia* (Fig. 181).

The anterior lobe is of ectodermic origin, is glandular in character and is developed from the primitive buccal cavity. It contains cells which are arranged in the form of convoluted tubes resting on a basement membrane. The blood supply is rich.

The posterior lobe, like the anterior is supplied by a connective-tissue capsule which sends tuberculæ into its substance. In the adult it consists mainly of neuroglia

with a few rudimentary ganglion cells and nerve fibers. In the human embryo the nerve elements are much more prominent.

The middle lobe or pars intermedia contains a connective-tissue framework and epithelial cells. It is less vascular and its cells are smaller than those of the anterior lobe. The pars intermedia contains also small cyst-like structures which contain a colloid substance something like that of the thyroid gland.

The functions of the pituitary body as a whole and of its different parts, are described under the head of "Acromegaly." Its disease produces conditions which go under the names of hyper-pituitarism and hypo-pituitarism and dys-pituitarism. Acromegaly, giantism, disturbances of sexual function and of carbohydrate metabolism are among the disorders attributed to this organ, destruction of it not being compatible with life.

The pineal gland or epiphysis cerebri is a small organ about the size of a cherry stone placed between the posterior ends of the two thalami and attached by a hollow pedun-

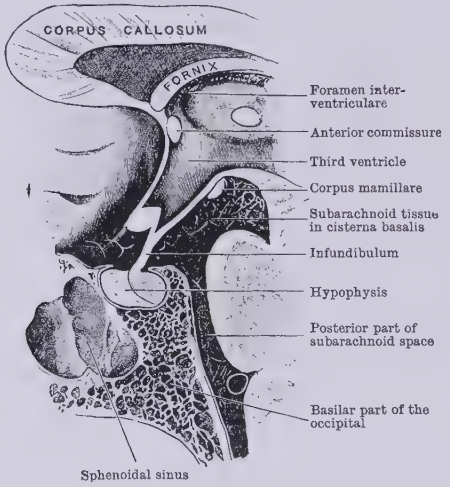


FIG. 181.—Mesial section, showing pituitary gland and connections. (*Cunningham.*)

cle to the dorsal aspect of the mesencephalon. It lies quite near the anterior opening of the aqueduct of Sylvius, and its position is such that it is possible in certain malpositions that it may occlude this aqueduct. It is a vestigial organ and is the representative of a median eye of invertebrates, developed from the second neuromere. It has no special sense function in vertebrates except possibly in the larval forms of the very lowest types. It is surrounded by a connective tissue capsule which sends trabeculæ into the organ dividing it into small chambers. These contain tubules lined with cuboidal epithelium (Fig. 182). According to Krabbe, the pineal gland contains parenchymatous cells which he terms special pineal cells. There are a few neuroglia cells, but no nerve fibres except those that go with blood-vessels.

The general statement has been that this gland degenerates at the seventh year. Krabbe has studied the anatomy of the pineal gland in children and in 100 adults. He finds no evidences of degeneration in the gland in adults and states that the structure in a man aged ninety-two was similar to that of a child of fourteen. He describes the parenchymatous cells and believes them to be secretory in function.

Investigations made by the writer with Dr. Berkeley, and the clinical and pathological observations made by a number of observers tend to show very strongly that the gland has secretory function, having to do with the processes of nutrition and growth. Its over-activity apparently produces increase in sexual, mental and somatic development. (See "Acromegaly.")

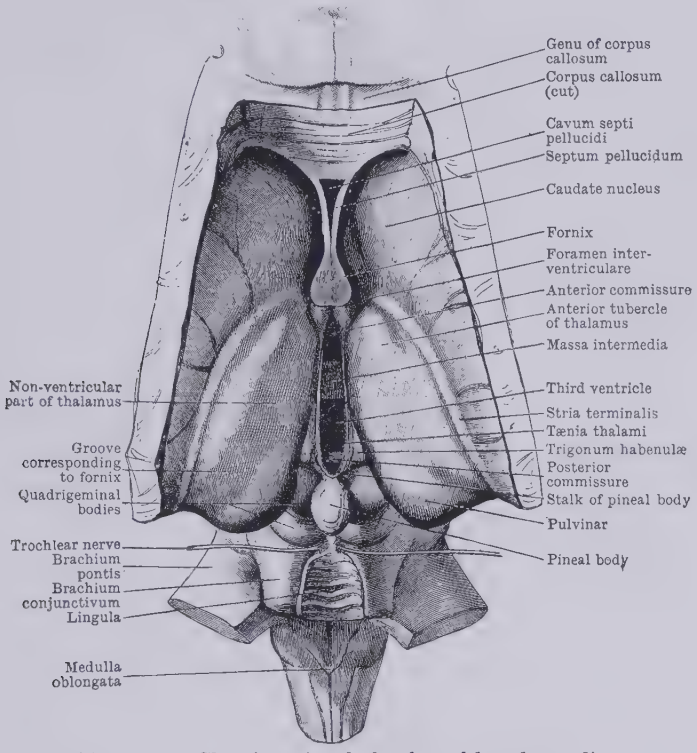


FIG. 182.—Showing pineal gland, and basal ganglia.

THE CONNECTING TRACTS OF THE BRAIN

The white matter of the brain, as already shown, is made up of:

1. Association fibres { Short. Long. Commissural.
2. Projection fibres and interganglionic tracts.

1. The association fibres of the cortex have been already described.

2. The *projection fibres* are those which connect different areas of the cortex with the basal ganglia and the ganglionic masses of the pons, medulla and spinal cord.

The projection fibres of the brain form the different pathways (*a*) by which the special and general sensations pass up to the brain cortex and (*b*) by which the voluntary, automatic, and psycho-reflex movements of the body are brought about.

The Efferent Tracts.—The cerebrospinal efferent paths are of two kinds: The direct or voluntary and the indirect or extra-pyramidal tracts.

The direct motor or upper motor neuron tract originates from cells in the precentral

convolutions; the neuraxons of these cells pass down and are gathered together in a narrow band which passes through and occupies the anterior two-thirds of the posterior segment of the internal capsule. The fibres continue on into the pons Varolii and medulla, and at the latter point give off some terminals which cross (except those for the sixth nerve) to the nuclei of the motor cranial nerves. The rest of the bundle passes on through the medulla, and 90 per cent. cross over at its lower portion,

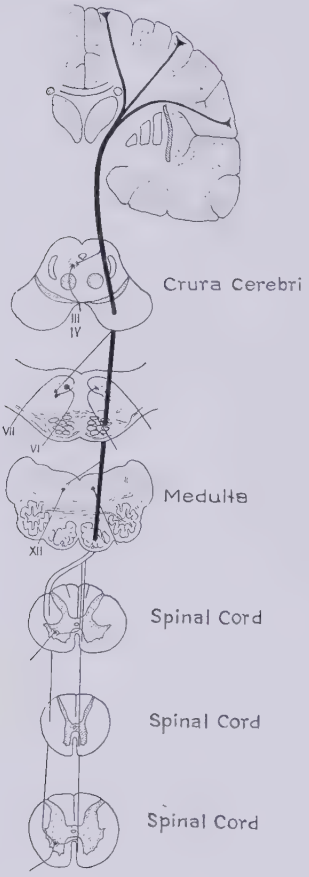


Fig. 183.

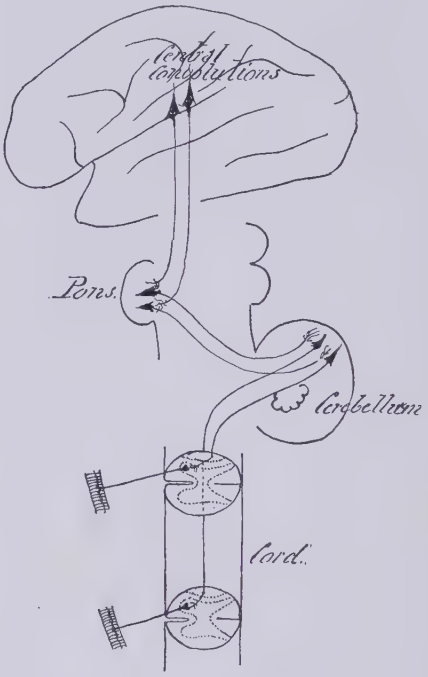


Fig. 184.

Fig. 183.—Showing the course of the pyramidal (cortico-bulbo-spinal motor) tract. Showing the upper and the beginning lower motor neurons.

Fig. 184.—Diagram of the indirect motor tract.

forming there the anterior pyramids. About 10 per cent. of the fibres do not cross, however, but continue on the same side. The crossed bundle passes into the lateral column of the spinal cord, forming the crossed pyramidal tract, which passes on, diminishing in size as far as the sacral part of the cord. It gives off terminal end brushes which surround the cells of the anterior horns. The small uncrossed band of fibres continues on in the mesial part of the anterior column, forming the direct pyramidal tract, or column of Türck. The fibres of this tract cross over in the anterior commis-

sure at different levels, and their teminals also connect with the motor cells of the anterior horns. From these horns the lower motor neuron passes out to the muscles. Hence the voluntary motor system is a two neuron mechanism.

Each tract as it reaches the spinal cord contains about 80,000 fibres.

The indirect efferent tract (*long extra-pyramidal tracts*) arises from nerve-cells in the anterior central convolution and, to some extent in the frontal lobe adjoining. Its fibres pass down into the internal capsule, mingling directly with those of the direct motor tract and giving off collaterals to the optic thalamus. The fibres pass through the cerebral peduncles, occupying their inner four-fifths, or motor part, and, finally, reach certain deposits of nerve-cells in the pons Varolii known as the pons nuclei. They surround these cells here with terminal end brushes. From these cells neuraxons cross the median line in the middle cerebellar peduncle and thence to the cortex of the lateral lobes of the cerebellum, where they in turn end. From here the nerve-cells send fibres to the nuclei of the cerebellum and Deiter's nucleus, thence down into the spinal cord, where they pass along mainly in the lateral fundamental columns, to connect finally with the anterior-horn cells. Other fibres from the cerebellar cortex pass to the dental nuclei, then cross to the red nuclei. Then some fibres pass spinalward in the lateral columns to the anterior horns. Thus it will be seen that the indirect motor tract is composed of (*a*) a cortico-pontile neuron, (*b*) a pons-cerebellar neuron, (*c*) a cerebello-nuclear neuron, (*d*) nuclear-rubro-spinal and nuclear-Deiter-spinal, and (*e*) the peripheral motor neuron.

The short extra-pyramidal tracts have been described (see p. 202), but they may all be grouped together here. They all end in the cells of the anterior or lateral horns of the cord.

SUMMARY: EFFERENT TRACTS FROM BRAIN TO CORD

2 Neuron system from cerebellum.	I. *Pyramidal tract:* (1) cortex of anterior central convolution. (2) Spinal cord to muscle.
2 Neuron system from mid-brain.	II. *Short extra-pyramidal tracts.*
	A. Tecto-spinal tract: (1) corpora-quadrigemina crossing to anterior horn cells, (2) to muscle.
2 Neuron system from mid-brain.	B. Fasciculo-spinal tract: (1) Cajal's nucleus in posterior longitudinal bundle, uncrossed to anterior column, to anterior horn cells, (2) to muscle.
2 Neuron system from mid-brain.	C. Rubro-spinal tract: (1) Red nucleus, crossing to antero-lateral tract, to anterior horn cells, (2) to muscle.
2 Neuron system from after-brain.	D. Vestibulo-spinal tract: (1) Deiter's nucleus crossing in part to peripheral part of antero-lateral column to anterior horn cells, (2) to muscle.
3 Neuron system from vestibular nerve.	
4, 5, or 6 Neuron system.	III. *Long extra-pyramidal tract:* Cortico-pontile; pons-cerebellar; cerebellar-nuclear; nuclear-Deiter; Deiter-spinal; anterior horn cells to muscles.

The direct or pyramidal tracts are all concerned with voluntary motion and inhibition, and when the anterior-horn cells of the cord are cut off by a lesion of these

tracts there is a spastic or upper neuron type of paralysis. The indirect and extra-pyramidal motor tracts are concerned in the co-ordination of bodily movement and in securing muscular tonus and synergy and in the higher reflex automatic acts. The course of the direct and indirect motor tracts is shown in the accompanying diagrams (Figs. 183, 184).

The Afferent Tracts.—The next important pathways in the brain and cord are the sensory, and they are concerned in bringing tactile, muscular and general sensations from the remoter parts of the body to the cortex of the brain. It will be easier

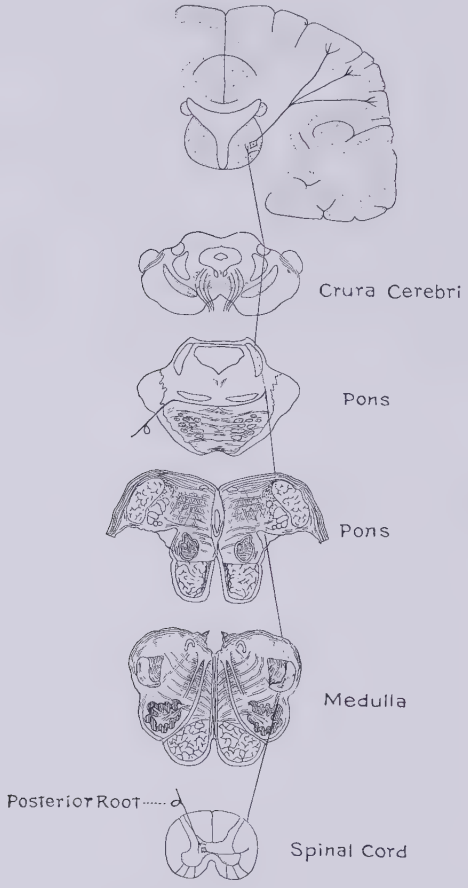


Fig. 185.—The afferent tract (3 neurons) for pain and temperature.

to follow these tracts if we begin at the periphery and follow the course of the fibres up to their centres in the brain.

The Cutaneous Sensory Tract.—A tactile irritation of the skin passes up the sensory nerve to a posterior spinal ganglion, where the cell body from which the fibre is derived is found. It passes directly through the ganglion, enters the posterior root of the spinal cord, and passes up to a group of cells lying in the posterior horns, where it meets and surrounds with its end brush a second sensory cell. A second neuraxon starts from the body of this cell; most fibres cross over in the posterior commissures

of the spinal cord to the posterior column of the other side where they run up in the posterior columns and gray matter (probably in chains of successive neurons), reach the medulla and pons, and pass through this to the optic thalamus. Here they send terminals to another cell, which in turn sends a neuraxon to the cortex of the post-central convolutions. Thus the cortical centres of the direct sensory path are just posterior to the area from which the motor tract started. The direct tactile tract is made up of (1) a peripheral sensory neuron, (2) a series of intraspinal neurons, (3) a spinal-thalamic neuron, (4) a thalamic-cortical neuron

The pain and temperature fibres take a similar course except that they cross over in the anterior commissure, do not run in chains of neurons and ascend in the lateral columns to the thalamus (Fig. 185).

The deep sensory tracts convey impulses which originate in muscles, joints and the viscera. The impulses pass up sensory nerves through the posterior roots. Some now pass directly into the posterior column of the cord of the same side and ascend till they reach the upper end, where their end brushes surround the cells of the nuclei of the column of Goll and of Burdach. From there they cross to the other side in the sensory decussation. Some then go to the red nuclei and optic thalamus, where they terminate. Another neuron now carries the impulse on to the parietal convolutions. Another set of deep afferent impulses goes from the sensory roots to the cells of the column of Clark, thence by the cerebellar tracts to the cerebellum. This indirect afferent tract is thus composed of (1) a peripheral sensory neuron, (2) a spinal-cerebellar neuron, (3) a cerebellar-thalamic, and (4) a thalamic-cortex neuron. Here, too, the mechanism is not so simple. Some of the afferent fibres do not go higher than the cerebellum, but are reflected to lower nuclei of the brain stem and thus automatic movements and the higher reflexes are brought about. The direct sensory tracts carry, for the most part, the sense of touch, pain, and temperature. Some direct sensory tracts are concerned with the sensation from the muscles and joints which have to do with co-ordination, and also with visceral sensations. It is through the indirect sensory and indirect motor tracts that the automatic and psycho-reflex acts are performed.

SUMMARY: AFFERENT TRACTS FROM SPINAL CORD TO BRAIN

3 Neuron system. Deep sensibility.	I. (1) Posterior ganglion cell to posterior column and nuclei of columns of Goll and Burdach, (2) crossing in medulla to optic thalamus, (3) to cortex of parietal lobe.
3 Neuron system. Pain and temperature.	II. (1) Posterior ganglion cell to posterior horn, (2) crossing to antero-lateral column to thalamus, (3) to cortex of parietal lobe.
4 Neuron system. Tactile sensation.	III. (1) Posterior ganglion cell to posterior horn, (2) partly crossing to chains of neurons in gray matter, (3) posterior and lateral columns to thalamus, (4) to cortex of parietal lobe.
2 Neuron system. Equilibrium, etc.	IV. (1) Posterior ganglion to cells of Clark's columns, (2) uncrossed to direct cerebellar tract to cortex of vermis of cerebellum.
3 Neuron system. Equilibrium, etc.	V. (1) Posterior ganglion to cells of intermediate gray matter of cord, (2) partly crossing to ventral cerebellar (Gowers') tract, (3) to anterior cerebellar peduncles to vermis.

Other Projection Systems.—The optic, acoustic, and olfactory projection tracts are described in connection with their peripheral nerves or end organs.

THE MEMBRANES OF THE BRAIN

The membranes of the brain are the dura mater, the arachnoid, and pia mater. The dura mater lines the inner surface of the skull. It is attached loosely to the concavity, but closely to the base. It splits into two layers to form the venous sinuses of the skull. The inner of the two layers at certain points projects inward to form membranous septa. These are known as the great longitudinal or cerebral falx, the lesser longitudinal or cerebellar falx, and the tentorium. Hence both venous sinuses and membranous septa are formed out of the inner layer. The outer layer forms the periosteum of the bone. The dura mater is supplied with sensory nerves, chiefly by the trigeminus but posteriorly by the vagus. The blood-supply will be described later.

The arachnoid is a thin, transparent, fibrous, non-vascular membrane lying between the pia and dura and continuous with the spinal arachnoid. It bridges over the fissures and the depressions at the base of the brain and forms between the pia and itself certain lacunæ or cisterns. These are the central lacuna found at the beginning of the fissure of Sylvius, the callosal, those of the transverse fissures and of the lateral aspect of the pons Varolii. The space between the dura and arachnoid is called the subdural or arachnoid cavity. It is lined with epithelium and resembles other serous cavities. The inner surface of the arachnoid is connected with the pia by numerous delicate fibrous processes. The space between these membranes is called the *subarachnoid space.* It communicates with the subdural space by means of the foramen of Magendie, which lies in the part of the arachnoid that passes over the pons and medulla, closing in the fourth ventricle; also by means of the lateral recesses of the fourth ventricle.

The subdural and subarachnoid spaces contain a serous fluid. It is called the cerebro-spinal fluid, and comes mostly from the choroid plexus of the lateral ventricles. The arachnoid contains no nerves or blood-vessels. It is described by some as a part of the pia mater.

The pia mater lies beneath the arachnoid and is closely applied to the brain in all its folds. It is continuous with the spinal pia. It is very vascular and supplies the whole periphery and part of the interior of the brain with blood. It consists of two layers: an outer holding the larger vessels, and an inner delicate layer closely associated with the superficial neuroglia of the brain. The pia mater folds upon itself and passes through the transverse fissure into the third and lateral ventricles of the brain. These vascular folds form the velum interpositum, which gives off a choroid plexus to the lateral and third ventricles. Another fold, the inferior choroid plexus, is given off to the fourth ventricle. The pia mater has vasomotor, but no sensory nerves.

Functions of the Brain Membrane.—The dura mater, by its outer layer, acts as a periosteum; by its inner layer as a lymph sac. It is also, by virtue of its sensitiveness, a protection against injury and disease. It is supplied with sensory nerves, but is not, in fact, very sensitive normally and may be cut and handled without pain. Strains upon it, however, do cause some pain (Cushing). The arachnoid forms the inner wall of the sac. The pia mater is a vascular and nutritive organ. It is, however, also closely connected with the lymphatic system of the arachnoid. The blood and cerebrospinal fluid of the brain vary in amount. In congestion the cerebrospinal fluid can pass into the spinal subarachnoid cavity or be rapidly taken up by the absorbents. In anæmia and atrophy there may be compensatory increase of the fluid.

The cerebrospinal fluid is a secretion and comes mostly from the choroid plexuses

of the lateral ventricles which are covered by the ependyma. These can, if needed, pour out an enormous amount which flows into the third and fourth ventricles and thence through the foramina of Magendie and Luschka into the subarachnoid cavities of the brain and cord. It flows under a pressure of 5 to 20 mm. of mercury and is eventually absorbed by the blood-vessels (veins and sinuses of the brain). A little of it probably leaks into the cavity of the arachnoid proper. The cerebrospinal fluid is colorless, transparent, alkaline. Specific gravity, 1,007.

Its composition is about as follows (Halliburton): water 99, solids, 1. The solids consist of: Proteids, 0.085; extractives, 0.060; salts, 0.865.

The proteids consist of serum globulin, and allied bodies. (Albumin is a normal ingredient, according to Bernstein.) A substance that reduces Fehling' solution is present. There is no fibrin ferment, and the fluid does not coagulate on boiling, or there is only a trace of turbidity. Under the microscope it is practically free from cellular bodies. The normal amount of fluid ranges from ʒii. to ℥ii., being greater in the aged.

There occur changes in the spinal fluid in inflammatory and degenerative diseases of the spinal cord, including syphilis, tumors of the cord and brain, and herpes zoster.

The Blood-supply of the Brain and its Membranes.—The vascular supply of the scalp, skull, and dura mater comes from the external carotids; that of the eye, brain, and pia mater from the internal carotids and vertebrals. The arrangement is shown here:

- External carotid gives off
  - Occipital, inferior meningeal, arteries.
  - Posterior auricular.
  - Temporal
    - Anterior.
    - Middle.
    - Posterior.
  - Ascending pharyngeal, posterior meningeal.
  - Internal maxillary, middle meningeal, small meningeal.
- Internal carotid gives off
  - Anterior meningeal.
  - Anterior cerebral.
  - Middle cerebral.
  - Posterior communicating.
  - Anterior choroid.
- Vertebral and basilar give off
  - Posterior meningeal.
  - Inferior cerebellar.
  - Anterior cerebellar.
  - Superior cerebellar.
  - Posterior cerebral.

The *blood-supply of the meninges* comes from the anterior, middle, and posterior meningeal arteries. These all come, except the small anterior meningeal branches and a small posterior branch, from the external carotid. The blood passes into the diploic veins, and from there passes chiefly into the lower occipital and lateral sinuses. Some of it, however, returns in the venæ comites. It all returns down toward the base of the skull. The most important of the arteries is the middle meningeal, both on account of its size and its distribution above important functional areas.

The *blood-supply to the pia mater and brain substance* comes from the internal carotid and the vertebral arteries. The branches of the former artery give off the anterior and middle cerebral, the posterior communicating and anterior choroid. The vertebral arteries give off the inferior cerebellar, while the basilar branch of the vertebrals gives off the transverse, anterior cerebellar, superior cerebellar, and posterior cerebral arteries.

The anterior, middle and posterior cerebral arteries, are the three largest and most important. By their anastomoses the circle of Willis is formed (Fig. 161). From the circle of Willis and the beginnings of the three arteries mentioned, several groups of vessels, six in all, are given off. They enter the base of the brain and supply the great basal ganglia and adjacent white matter. They are called the *central arteries;* they do not anastomose, and they are the vessels usually affected in cerebral hemorrhages of adult life. One set of these central arteries, the *internal striate,* passes up and supplies the two inner segments of the lenticular nucleus, the caudate nucleus and anterior part of the internal capsule. Another set, the *external striate,* divide into anterior or lenticulo-striate branches which supply the lenticular nucleus and posterior

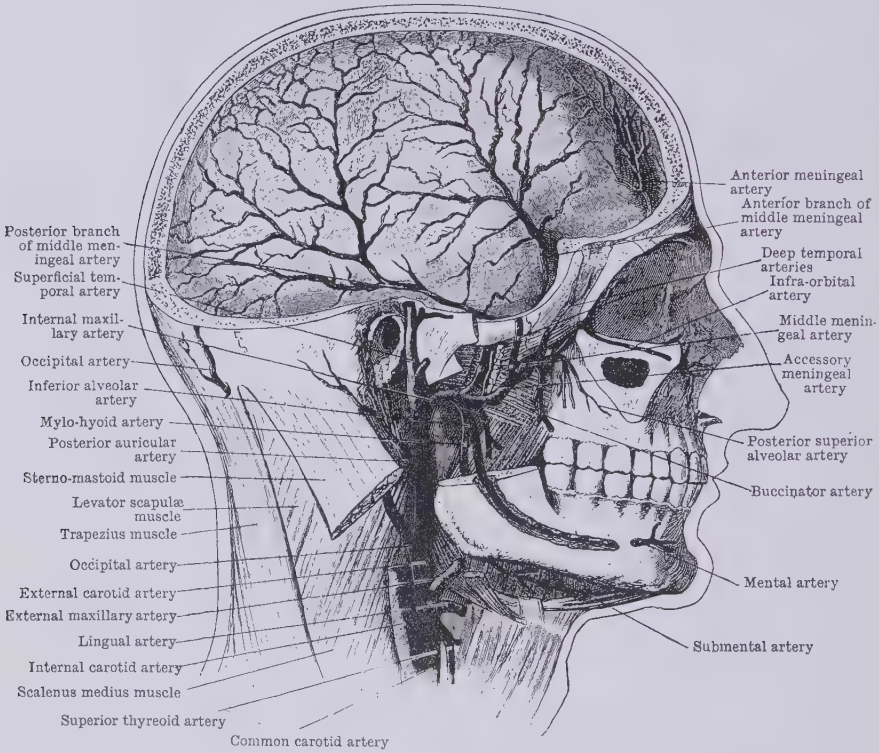


FIG. 186.—The external carotid, internal maxillary and meningeal arteries. (*Cunningham.*)

part of the caudate. One of these larger than the rest is called the artery of cerebral hemorrhage. The other or posterior branch is called the lenticulo-optic and supplies the anterior part of the thalamus. According to Kalisko, the lenticular nucleus is supplied in part by a branch from the anterior cerebral. This he calls the artery of cerebral thrombosis. It is especially effected in severe gas poisoning.

The *cortical arteries* are the terminal branches of the great cerebral arteries. They anastomose with each other but slightly They are distributed very widely and carry much more blood than the central groups. Their distribution is shown in Figs. 187, 188. The cortical arteries are distributed in the pia, and from there they pass in two sets, a superficial and a deep, into the gray matter, and for a short distance into the white matter. They pass straight in at right angles to the surface. They

have richly arborescent branches which do not anastomose; consequently a knife plunged straight into the brain does not cut many vessels.

The capillaries are surrounded by spaces called perivascular spaces which serve as lymphatic channels. The neuroglia cells send processes which connect with or form passages to the vessel walls.

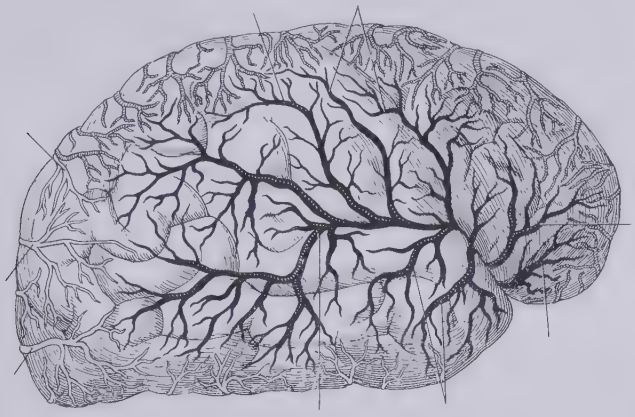


FIG. 187.—Distribution of arteries on convexity of cerebrum. Middle cerebral in black; anterior cerebral in gray; posterior cerebral in white.

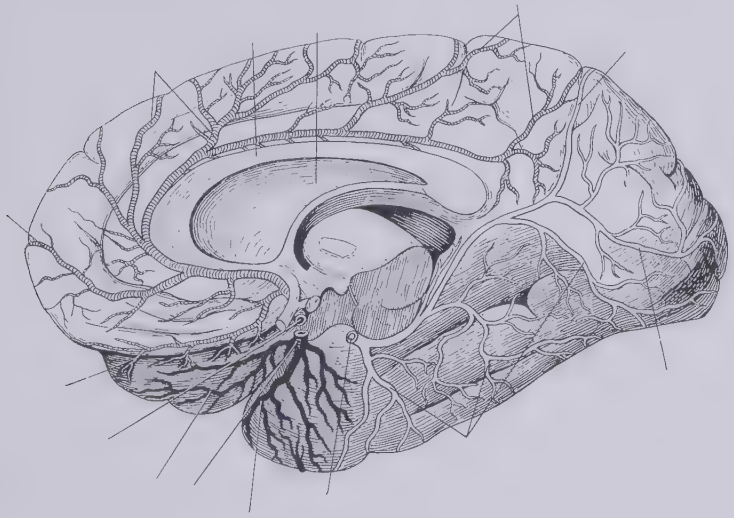


FIG. 188.—Arteries of mesial and inferior surface of cerebral, in black, gray and white as in previous figure.

NOTE.—The *caudate nucleus* is supplied by the anterior and middle cerebral central arteries.

The *lenticular nucleus* by the anterior choroid (part of its internal and external segments), by the anterior cerebral (caudate nucleus and external and middle segments of lenticular), by the middle cerebral (caudate nucleus and middle and external segments of lenticular).

The *optic thalamus* by the posterior communicating anterior choroid (external nucleus) and the posterior cerebral. (Fig. 209, p. 434.)

The *corpora quadrigemina* and geniculate bodies are supplied by the posterior cerebral.

The *red nucleus* by the posterior cerebral, and the *subthalamic region* by the posterior communicating.

The *internal capsule* by the anterior cerebral and middle cerebral for its anterior part, by the posterior communicating, anterior choroid and the middle cerebral for its posterior part. None of the thalamus is supplied by the middle cerebral and none of the internal capsule by the posterior cerebral. All direct thalamus lesions must be posterior cerebral, or in minor degree posterior communicating. No direct capsular lesions would come from the posterior cerebral (Beevor). (See, however, Fig. 209, Bing.)

The cortical areas of the brain supplied by the anterior, middle and posterior vary somewhat; the three chief systems anastomose at the parts where one region joins the other (Duret, Beevor). The cortical arteries do not anastomose with the central or ganglionic arteries which latter supply the centrum ovale.

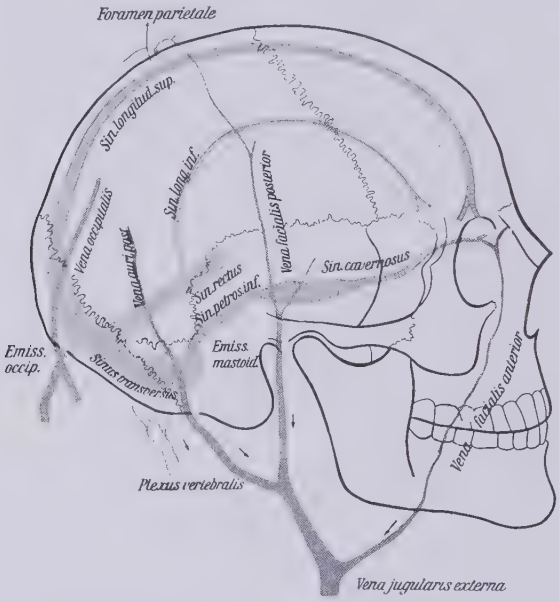


Fig. 189.—The cerebral sinuses. (*Hermann.*)

The blood of the convex and mesial cerebral surface, flowing up from the base, leaves the capillaries and enters veins. Thence it still passes upward, and for the most part enters the longitudinal sinus. The most of the vessels enter the posterior portion of the sinus and in a direction forward and upward; *i.e.*, against the current in the sinus. The course of the blood-current is, therefore, opposed both to gravitation and to the venous flow.

The *veins of the cerebrum* are: 1, the superficial cerebral; 2, the deep cerebral and, 3, the cerebral sinuses. 1. The superficial cerebral veins are venæ comites. Those on the convex and mesial surfaces empty chiefly into the superior longitudinal sinus, as described; those on the basal surface and temporal lobes empty into the cavernous and lateral sinuses. These veins have no valves, and their walls are very thin and without muscular fibres. 2. The deep cerebral veins, or venæ Galeni, receive the blood from the lateral ventricles and from some of the central arteries

supplying the basal ganglia. They empty into the straight sinus. These deep or central veins when compressed lead to stasis and internal hydrocephalus. 3. The *cerebral sinuses* are fifteen in number. The important ones are the superior and inferior longitudinal, the straight, the lateral, the occipital, the cavernous, and the superior and inferior petrosal. They carry blood for the most part in a direction from before backward, and convey it eventually to the internal jugular (Fig. 189).

Most of the blood of the convexity and mesial surface must pass into the longitudinal sinus, but there is a slight connection of some of the veins with the superior petrosal and straight sinuses. The superior longitudinal sinus also communicates slightly with veins of the scalp and with the facial vein. Some of the blood from the mesial surface also goes to the veins of Galen.

On the whole, however, the system of the convex and mesial cerebral surface is a close corporation, the blood having to pass into the superior longitudinal sinus and

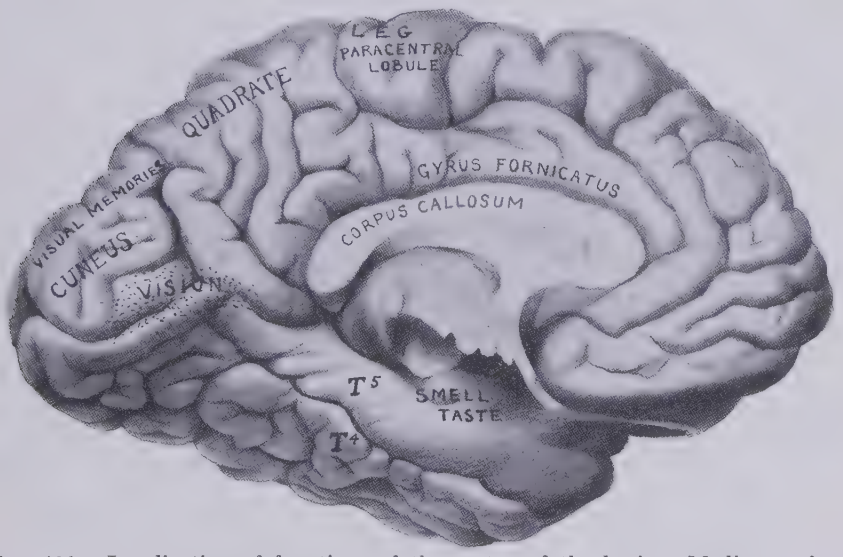


FIG. 190.—Localization of functions of the cortex of the brain. Median surface.

torcular Herophili, where it meets that of the straight and occipital sinuses, and flows forward through the lateral sinuses to the internal jugular. The circulation of the basal surface is less isolated. All the basal sinuses communicate with each other freely, and there are slight communications between the veins of the scalp and the cavernous, lateral, and inferior petrosal sinuses. It is safe to tie any of the sinuses, except the lateral and the posterior part of the longitudinal. The cerebellar veins, superior, inferior, and lateral, empty into the straight, the lateral, and superior petrosal sinuses. None of the cerebral veins or sinuses have valves.

The pressure of the blood as it goes through the external carotid is about 150 to 200 mm. of mercury. The pressure of the blood in the cranial cavity is much less (from 0 to 50 mm. of Hgr., Hill). The pressure in the central arteries is greater than that of the cortical. The pressure of the venous blood is very slight and is ordinarily the same as that of the cerebrospinal fluid.

Both arteries and veins are more delicate than the extra-cerebral vessels.

Except in gray matter, the brain is not a very vascular organ, but this gray tissue

ranks in richness of blood-supply with the lungs and liver. The amount of blood in the brain at any one time is only about 1 to 2 per cent. of the total blood in the circulation, or about four ounces (Ranke).

THE FUNCTIONS OF THE BRAIN

Cerebral Localization.—The brain is the seat of conscious intelligence and other psychic activities. It exerts control and direction over voluntary movements, it is the seat of instinctive acts, and it regulates in a measure the vaso-motor, trophic, and secretory mechanisms of the body.

The Prefrontal Lobes.—The prefrontal lobes, or that part of them which is in front of the precentral convolution, are concerned with the higher association processes and memories. The most anterior portion, according to Campbell, though of late development is poor in cortical cells. In the posterior part are centres for movements

FIG. 191.—Same. Superior surface.

of the head and eyes, and in the left third frontal is a centre for speech or articulation memories. Injuries of the prefrontal generally lead to changes of character, indicated by peevishness and irritability of temper, mental enfeeblement, lack of power to concentrate the mind or to control the acts or emotions. The left lobe is said to be more important psychically.

The Central Convolutions.—This part of the brain is called the *sensori-motor area*, because it is concerned in the receipt and emission of nervous impulses which lead to voluntary motions of the body. Certain parts of this area are in relation with certain groups of voluntary muscles on the opposite side of the body. These areas preside not so much over single muscles as over those groups of muscles which act together in producing definite, purposeful acts. The motor area proper is confined to a narrow

space on the posterior surface of the precentral convolution extending down to the bottom of the fissure of Rolando. It is co-extensive with deposits of the giant cells of Betz. (Sherrington and Greenbaum). The lower part of the central convolutions, known as the central operculum, is a centre for movements of the larynx, mouth, tongue and face. This area reaches to the lower bend or genu of the fissure of Rolando, near which is the centre for the neck. Above and occupying the middle third of the convolution are centres for the shoulder, arm, hand and fingers, which extend up as far as the upper genu, near which is the centre for the trunk. Above this genu are centres for the hip, leg, toes, the area extending over into the paracentral lobules. Near the base of the first and second frontal convolutions is a centre for movements of the head and eyes. The exact arrangement of these centres, which has been determined by experiments upon monkeys and other lower animals as well as by clinical and surgical observations on man, is shown in the accompanying Figs. 190, 191, 192. The posterior central convolution is probably the centre for cutaneous sensations.

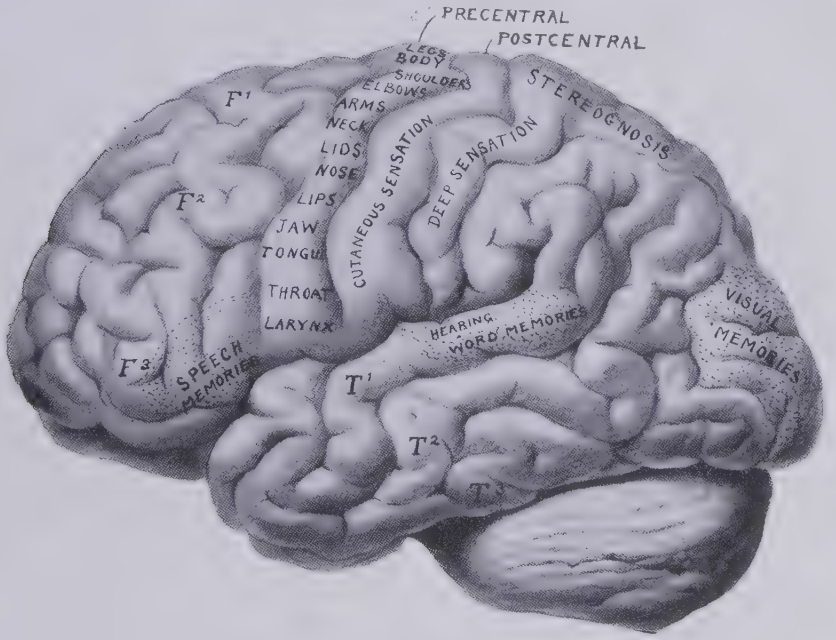


FIG. 192.—Localization of the functions of the brain. Lateral surface.

The various sensori-motor centres are not sharply limited, but lap one over the other, so that the motor area for the arm, for example, extends over somewhat into that for the shoulder. The corresponding sensory areas are more diffuse, so that it takes a much more extensive destruction of a certain area of the cortex to produce an anæsthesia of the arm than it does to produce a paralysis of the arm. The strictly motor and sensory areas have closely adjacent to them psychic-motor and psychic-sensory areas, concerned in the higher elaboration of movements, sensations and reflexes.

Bilateral Representation.—Those muscles of the two sides of the body which act together have a double representation in the brain. For example, each group of muscles used in inspiration has a centre in both hemispheres; consequently, when one

centre is destroyed no paralysis results, for the reason that the other centre continues its work. In the same way some of the muscles of the face, such as those for closing the eyes, have a double representation, and a lesion destroying the centre for the orbicularis palpebrarum on one side will not usually cause paralysis, because of the continued action of the centre of the other side. The more perfect and habitual the associated action of the muscles of the two sides of the body, the more completely can one centre do the work of its associate. The best examples of the muscles having the double representation are the orbicularis palpebrarum, the muscles of the vocal cords, the muscles concerned in deglutition and in respiration. The muscles of the viscera and blood-vessels have no known representation in the cortex of the human brain.

The special sensations have a bilateral representation also; but the more specialized the sense the less can one hemisphere take the place of the other.

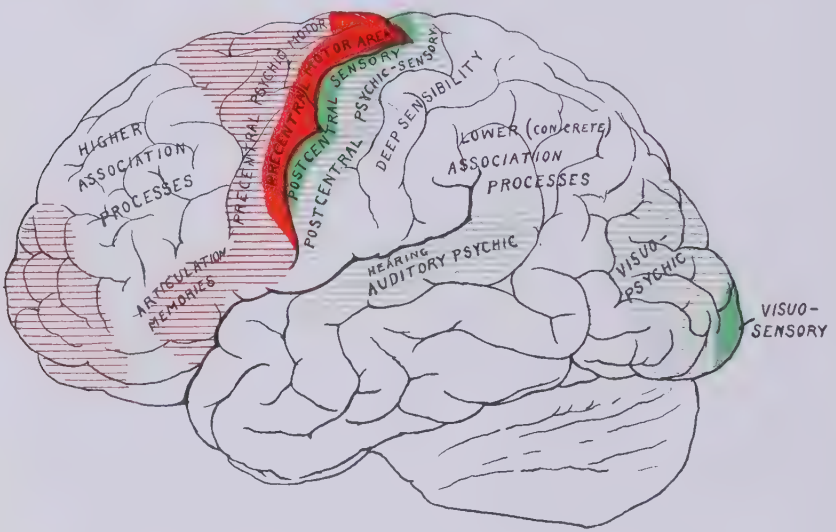


FIG. 193.—Localization of the functions of the brain cortex, showing the psychic areas. (*Campbell.*) Lateral surface.

Parietal Lobes.—The upper portion of the parietal lobe is the centre or region, for we include both the cortex and its underlying tracts, for association of memories of form, or more properly the perception of the form. When destroyed there is astereognosis or loss of perception of form; and if with it there is some involvement of the zone of language, there is asymboly; that is, an inability to tell what the object is even if the form is recognized. Lying anteriorly in the lower part of the parietal convolution below the parietal fissure, is an area for memories of muscular movements, and when this is injured there is ataxia, or loss of sense of position. The whole parietal lobe seems to have to do with association processes connected with the elaboration of sensations. It is a percipient or, as Mills calls it, a concrete memory centre, not a sensory centre. Even the ataxia which occurs here is a loss of perception, and not a pure sensory loss.

In its posterior and inferior part, on the left side, we have part of the zone of language, particularly that concerned in memories of written words (reading centre). Near this is, perhaps, a centre for memories of the uses of things (praxic centre).

Occipital and Temporal Lobes—*Centres of Special Sense.*—The special senses have two centres—the primary and the secondary. The primary centres are connected with the ganglia at the base of the brain; the secondary centres are situated in the cortex.

The *primary centre for vision* is in the posterior part of the optic thalamus, the external geniculate bodies, and anterior corpora quadrigemina. The secondary centre is situated in the occipital lobe, and particularly upon its mesial surface and

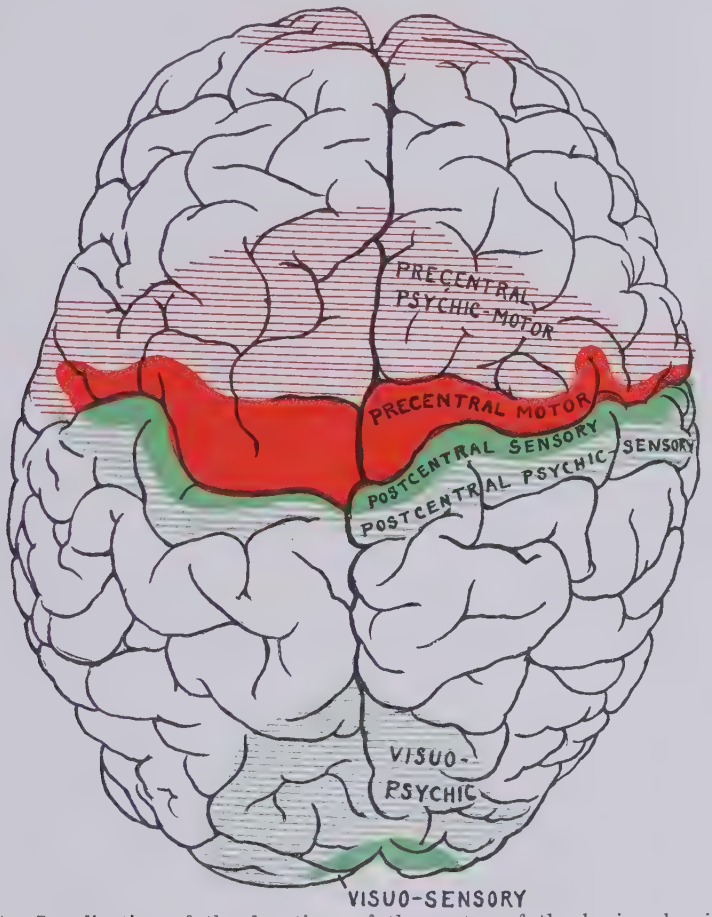


FIG. 194.—Localization of the functions of the cortex of the brain, showing the psychic areas. (*Campbell.*) Superior surface.

about that part of the cuneus known as the calcarine fissure. Each occipital lobe is the centre for visual impulses from the corresponding half of the retina of each eye; for example, the left occipital lobe is the centre for vision of the left half of the retina of each eye. This relation is shown in the diagram (see Optic Nerve). Total destruction of both occipital lobes, or even of a considerable part of them if the destruction involves the median surface, will cause blindness. Destruction of one lobe causes only half-blindness or hemianopsia.

The *primary centre for hearing* is in the posterior tubercles of the corpora quandrigemina and the internal geniculate bodies. The secondary centre is in the cortex of the first, including the transverse convolution of the temporal lobe. Destruction of one temporal lobe causes some deafness in the opposite ear. This deafness, however, is not complete because the sense of hearing has a bilateral representation; each ear in other words, sends fibres to the temporal lobes of each side, although more fibres cross over than go to the lobe of the corresponding side. The consequence is that the loss of one temporal lobe is in a measure supplied by the other (see also Aphasia).

The *primary centre for smell* is in the olfactory lobes. The secondary centre is probably in the anterior part of the limbic lobe, the uncus and in part of the hippocampal convolution. Whether the tracts for the sense of smell are connected with the optic thalamus or other ganglia is not definitely known.

The primary centre for taste is not known, but sensations of taste may connect

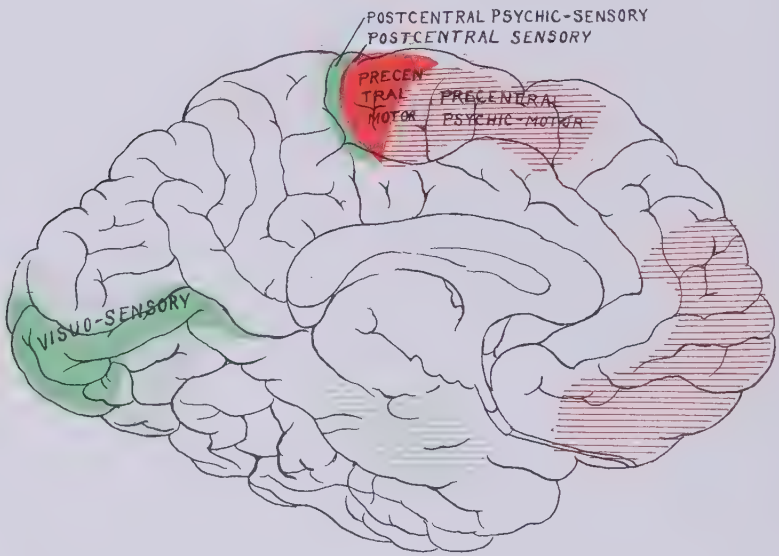


Fig. 195.—Same. Median surface.

with the optic thalamus before passing into the secondary centre, which is in the hippocampal convolution.

Centres for Special Associations.—There are certain sensations, perceptions and movements, simple in character and frequently repeated, so that they finally get to be used almost automatically in their work. These impressions relate to the use of the muscles in speech, in writing and in gesture language; also to other frequently repeated purposeful movements of the limbs. The muscular movements in writing and speaking are so often repeated that certain areas in the cortex are set apart for the memories of these processes, memory being simply a revival and re-association of previously registered impressions. The visual sensations and the ideas elaborated from them, which are frequently repeated in learning to read, have also a centre which is set apart for them. This forms a centre for the memories of written language. In the same way there are auditory sensations and ideas elaborated and so frequently repeated as to be used automatically in acquiring language. These are stored up as

auditory memories. We have what may be called motor memories connected with speech and gesture. These special memories have been found to have a certain localization in the brain. The centre for the memories of the articular movements of speech is in the posterior part of the third left frontal convolution; the centre for the memories of the movements of writing is not perfectly known, but is thought to be at the posterior part of the second left frontal convolution. The centre for the memories of gesture language is unknown. The centre for the memories of ordinary co-ordinate movements is probably in the inferior parietal lobule. The centre for the visual memories of written language is in the angular gyrus, extending backward from there into the occipital lobe. The centre for the audtiory memories of spoken language is in the posterior part of the first and the corresponding upper part of the second temporal convolution. In right-handed people the language memory centres are in the left cerebral hemisphere; in left-handed people they are in the right hemisphere. The centres for the memories and associations connected with language together form a large area, known as the zone of language. The destruction of these memory centres produces different forms of aphasia, as will be described later.

In addition to, disturbances in centres are produced by remote lesions which cut off the associating fibres connecting these centres with each other or with motor or sensory centres proper, causing a phenomenon called diaschesis.

The Centrum Ovale, Corpus Callosum, and the Associative Functions of the Brain.—The different parts and centres of the brain are connected together by the associating tracts and with lower levels by the projection fibres. The simpler and less developed centres of the two halves of the brain are closely connected by fibres that run chiefly in the corpus callosum. The more highly specialized and less simple in function a centre, the less close is its commissural connection and the more independent is one half of the brain from the other. Thus the centres for the movements of the thorax in respiration are closely bound with each other; those for the purposeful movements of the hands less so; those for receiving visual impressions are almost independent; and the centres for language associations which are still more highly specialized, are practically entirely independent. We infer that the higher mental functions, therefore, work either in one cerebral hemisphere or in the other, and that the two halves of the brain do not co-operate with each other in much of the higher intellectual work. This, however, may not be true for the very highest mental processes, though persons have lived and shown fair intelligence without a callosum.

The corpus callosum is the great commissural tract connecting the two cerebral hemispheres and their respective centres. The anterior commissure does some of the same work, being more specially connected with the function of olfaction. The posterior commissure has comparatively few bilateral connecting fibres, its function being more to connect the thalamus with the cranial nerve nuclei and other centres below.

The Corpus Striatum.—This ganglion is in relation with the cerebral cortex slightly, with the internal capsule slightly, with the thalamus and subthalamic nuclei, and probably through these with the cerebellum and with nuclei in the pons. It is also in connection with fibres that come up from the muscle-sense tract, in the spinal cord. It seems most intimately connected with the thalamus and subthalamic ganglia. Its functions are therefore probably connected with supplementing automatic movements. Destruction, of this ganglion in the human brain produces at times motor and vasomotor disturbances. Lesions of it seem to have to do indirectly with the production of tremor and rigidity.

The Thalamus Opticus.—The thalamus is in relation by its projection fibres with the frontal, parietal, occipital, and temporal cortex. It is in relation below with tracts for cutaneous and deep sensibility. The fibres that go to the occipital cortex

are connected with the optic tract, and have to do with the function of vision. The fibres that go to the temporal lobe are connected with the auditory tract, and have to do with the function of hearing. The optic thalami have a probable relation to the expression of emotions. Lesions of the posterior part of the thalamus will produce hemianopsia and central pain. Other than this, lesions of the optic thalamus produce no definite symptoms which enable us to make a local diagnosis, except through injury of neighboring parts. Disturbances of hearing have not certainly been traced to lesions in the thalamus. Lesions of the thalamus sometimes produce various forms of mobile spasm, but these are generally attributed to irritation of the fibres of the internal capsule, which go close to it. Hence, aside from pain, disturbances of vision and of deep sensibility, the optic thalamus also must be considered clinically a latent region.

The Corpora Quadrigemina.—The anterior tubercles of the corpora quadrigemina, together with the external geniculate bodies, form part of the primary centres of vision, The anterior tubercles, however, have to do chiefly with reflex movements of the pupil and the ciliary muscles. The posterior tubercles of the corpora quadrigemina and the internal geniculate body are connected with the auditory nerve, and have to do with reflex movements associated with hearing and space sensations. They also appear to receive some fibres from the cerebellum; their injury or disease produces some disturbances in equilibrium and possibly in hearing. Owing to the fact that the nuclei of the third nerves and the red nuclei lie beneath the corpora quadrigemina, lesions of these latter produce nystagmus and paralyses of the third nerve, disturbances in equilibrium, and forced movements. Lesions in this neighborhood sometimes cause somnolent and stuporous states.

The *red nuclei* are connected with the anterior cerebellar peduncles on the one hand and with the lenticular nucleus and optic thalamus and spinal cord on the other, and are concerned in securing muscular tone, synergy and co-ordination.

The Cerebellum.—It receives impulses from the cerebral hemispheres, and these impulses go via the pons to the cortex of the lateral lobes. It also receives impulses from the vestibular and to some extent from the other sensory cranial nerves. These impulses go to the cortex of the vermis.

The cerebellum also receives impulses from the spinal cord *via* the direct (dorsal) cerebellar tract and the column of Gowers (ventral cerebellar tract), but not from the posterior columns. These impulses go to the cortex of the vermis. The efferent tracts of the cerebellum are those to the cerebrum and those to the mid-brain, subthalamic region and spinal cord. The cerebello-cerebral fibres leave the cortex of the lateral lobe, go to the dentate nuclei, thence through the anterior cerebellar peduncles, crossing, in most part, to the red nucleus and thalamus, perhaps to the nucleus tecti, thence to the cortex of the brain. The cerebello-spinal fibres start from the cortex of the vermis, then to the roof and emboliform nuclei (intrinsic cerebellar nuclei), thence to the nucleus of Deiters mainly (paracerebellar nuclei of Horsley), thence to the spinal cord in the anterolateral tracts, and anterior cornua. Some cerebello-spinal fibres go from the red nucleus and tectal nucleus to the cord. Hence we have vestibular-spinal, the rubro-spinal and the tecto-spinal tracts conveying impulses down to the cord, originating mainly at least in the cerebellum. Horsley has shown that the cerebellar cortex always sends out its efferent impulses first to the various nuclei of the cerebellum. Stimulation of these causes tonic movements of the head. Stimulation of the paracerebellar nuclei (Deiter's,) in the pons also causes tonic movements of the body. It is when these intrinsic and paracerebellar nuclei are irritated that we get cerebellar fits. L. Hill and others have shown, as Jackson first averred, that the cerebellum causes tonic fits, the cerebral hemispheres clonic fits. There is a nervous circuit between the cerebrum, brain axis, cerebellum

and spinal cord. The cerebellum has thus the function of organizing the higher automatic and psycho-reflex movements, and through its relations with the eighth nerve and spino-cerebellar tracts of enabling us to keep our equilibrium and maintain our relations in space. It also gives tonus and synergy to the muscles. The *vermis* or median lobe is the part which in man is most important in doing this work. The *lateral lobes* are concerned in acquiring new movements and dexterities and in synergizing different segments of the body (neck, arms, legs). (See also Figs. 218. p. 462.)

The *pons Varolii* contains some of the cranial nerve nuclei and collections of nerve-cells which are connected with fibres from the cerebral cortex on the one hand and the cerebellum on the other. It also contains the long tracts of nerve-fibres that pass from the cerebrum down through into the medulla and spinal cord and transverse tracts of fibres which connect the two hemispheres of the cerebellum. Lesions in it cause disturbances in function of the cranial nerves and of the motor, sensory and commissural tracts.

The *medulla oblongata* contains centres of the cranial nerves, and in it also are various reflex and automatic centres controlling and regulating the vasomotor system, respiratory and cardiac rhythm, visceral movements and secretion.

The *olivary bodies* are connected with the cerebellum, basal ganglia, and with the spinal cord. When injured, disturbances of equilibrium and co-ordination occur.

The Latent Regions of the Brain.—There are certain parts of the cerebral cortex destruction and irritation of which produce no special and distinctive phenomena in man. These are the greater part of the temporal lobe of the right side and a portion of the temporal lobe on the left side. A part of the inferior parietal lobule also may be regarded as a latent region. The frontal lobe we have already spoken of as being concerned with certain mental functions, but lesions here often produce no symptoms, and may be to a certain extent regarded as latent.

Brain Size and Weight.—The length of the brain averages 160 to 170 mm. and its transverse diameter is 140 mm.

The female brain is usually shorter than the male.

The average weight of the male brain is 1,358 grams; that of the adult female, 1,245 grams.

The weight varies with age, sex, race, and intelligence, and with a number of other factors. The average weight of the brain at birth is 327.8 gm.; the brain grows rapidly until the age of four, then more slowly until the age of seven, then very slowly up to the age of sixteen to twenty. At about the age of forty-five in man and fifty in woman it begins to lose weight slowly, and at the age of eighty or over it has lost about 120 gm. (4 oz.). The brain of man weighs absolutely about 9 per cent. more than that of woman. Relative to the body weight, the brain weight of man is about 2 per cent.; that of woman is very little less. The sexual difference is extremely small. Brown finds that, after making all allowances, woman's brain weighs about one ounce less than man's.

The brain weighs more in the civilized races, and more in certain of the civilized races than others; the brains of English, German, and Scotch weigh more than those of French, Italian, and Russian. Some of the African and Australian tribes have the smallest brain, the average negro brain weighing 1,250 gm.

Caucasian race average brain weight is 1,335 grams.
Chinese race average brain weight is 1,332 grams.
Malay race average brain weight is 1.266 grams.
Negro race average brain weight is 1,244 grams.
Australian race average brain weight is 1,185 grams.

The brain weight of highly civilized nations is somewhat larger than that of the uncivilized, and cultural activity in a race rather slightly increases the cranial capacity. When a brain weighs less than 1,130 gm. in man or 990 gm. in woman, it is called a microcephalic brain; if the weight is above 1,490 gm. in man or 1,345 gm. in woman, it is called a megalocephalic brain.

Brain weight has a certain relation to intelligence, which is not, however, an absolute one. Among a hundred men of more than average intelligence, the percentage of large brains would be about 25, whereas the percentage of large brains among persons of ordinary or low intelligence would not be more than 4 or 5. In estimating the importance of brain weight, one must consider the height, the weight or volume of body, muscular mass, and superficial area; these are called the somatic factors. The following formula has been devised by Snell for estimating the mental power of different animals.

$$P = \frac{H}{Ks}$$

In this formula P represents the psychical factor or the amount of intelligence, H the brain weight, K the body weight, S for the somatic factor. The somatic factor has been estimated to be for mammals about 0.666. Applying this formula, we find that, expressed relatively the intelligence of man equals 0.87; woman, 0.86; the ape, 0.42; the rabbit, 0.59; the birds from 0.167 to 0.09.

The brain weight as compared with the body weight is not so great in man as it is in certain birds.

The weight of the brain in the adult male as compared with the spinal cord is greater, however, than in any other race. That is to say, the spinal cord in the human subject weighs approximately 2 per cent. of the brain. In the anthropoid apes it weighs 6 per cent.; in American mammals, from 23 to 47 per cent.

The relative weight of different parts of the brain is about as follows: frontal lobes, 28 per cent.; parietal lobes, 36 per cent.; occipital, 10 per cent.; temporal, 13 per cent.; island of Reil, 9 per cent.; pons, 1½ per cent. The cerebellum weighs about one-eighth as much as the cerebrum. The proportion of the gray to the white matter in adults is 60 to 40 (Vierordt).

CHAPTER XVII

DISEASES OF THE BRAIN AND ITS MEMBRANES

GENERAL SYMPTOMS

General symptoms are developed when the brain and its membranes are subjected to irritating influences. These influences may be of various kinds, and are best illustrated by the irritant which occurs in the early stages of a febrile infection like meningitis. Similarly, general symptoms may be caused by conditions which benumb and depress or paralyze the activities of the brain. These influences are either mechanical, such as those of brain pressure, or perhaps severely toxic such as occurs in the advanced stages of an inflammation. Perversions of brain activity in various directions partly irritant, partly paralyzing also may occur.

There are also disturbances of brain function due to irritative or destructive foci in the brain.

The general symptoms of *brain irritation* are headache, vertigo, vomiting photophobia, mental irritability, insomnia, peculiar feelings of fullness and pressure about the head, noises in the ears or in the head, tenderness about the scalp, and in severe cases rigidity of the neck, convulsive symptoms and delirium.

The symptoms of *depression* of the brain functions are often caused by an inflammatory exudate or serous effusion which compresses the brain and hence they are spoken of as symptoms of brain *compression*. These, as they progressively increase, are headache, mental dulness, restlessness, mental confusion, clouding of consciousness, delirium, disturbed sleep and finally stupor and coma. As these symptoms progress there occur a slowing of the pulse, increased blood-pressure, irregular respiration, and papillary œdema of the optic discs. Finally the coma deepens, the respiratory and bulbar centres become paralyzed; there is now a rapid pulse and lowered blood-pressure, paralysis of respiration and death.

The *focal symptoms* of brain disease depend upon the location of the particular lesion. If it is in the motor area there will be symptoms of spasmodic character or of a local paralysis. If it is in the speech area there will be symptoms of aphasia.

Besides the symptoms which are due to the local disease there may be general symptoms produced by the pathological condition. Thus,

while a tumor of the brain will cause perhaps only focal symptoms of irritation or pressure, a suppuration in the brain will produce general symptoms such as are associated with sepsis, viz., chills, irregular fever, mental hebetude, prostration, emaciation, and sweats.

The dominant focal symptoms are: local convulsions, hemiplegia, hemiataxia, hemianopsia, cerebellar ataxia and dyssynergy, cranial nerve palsies, and aphasia.

Among these symptoms there is one which deserves some preliminary general study, because it is caused by lesions of different kinds and occurs in different forms of diseases. This symptom which we wish particularly to study here is aphasia.

Aphasia.—Aphasia is a disorder of the faculty of language; and it has a number of varieties, in accordance with the particular part of the brain involved and the particular portion of the mechanism of this faculty that is injured. By the faculty of language we include the processes by which we hear, see, and appreciate the meaning of symbols of language; and express to others by voice, writing, or gesture the content of our minds. Language has, therefore, a receptive side and an emissive side.

We may have lesions in the brain which destroy that part of the language faculty concerned in our power of seeing and understanding written words or the gesture language. In reading understandingly one sees certain words; these words revive certain visual memories connected with past perceptions. Thus one sees the word "book;" this suggests to him past memories of form, color, tactile and other sensations associated with the past perceptions of books. There is a certain centre in the brain where these visual memories for letters and words are associated. When this centre is destroyed the memories are destroyed and the word "book" or any other written word conveys no meaning. The patient can spell out the letters, he can see the letters, but he cannot read any more than if he had never been taught. The condition is known as *alexia* or *word blindness*.

Again a person may have learned to associate certain gestures with definite ideas, as the motion of carrying a glass to the mouth suggests that of drinking, or the motions of using a knife and fork that of eating, or the motions of the deaf-and-dumb alphabet with certain words and ideas. These memories of gesture language are located in certain regions, and when they are destroyed the patient is no longer able to understand gestures or the sign language. This condition is known as *sign blindness*.

When a person is not able to understand the significance or uses of things about him, he is said to have *apraxia*. For example he sees a knife and names it but he does not know its use. He knows its use but he does not know how to make the movements necessary to put it to use. In the former case he has sensory apraxia (imperception, agnosia are

other terms). In the latter case he has motor apraxia (also a form of agnosia).

A person hears certain words, as, for example, the word "knife." This conveys to him a certain idea of the form, color, and other properties associated with knife. The memories associated with the auditory perception of different words are stored up in a certain locality which is the centre for auditory memories. When this centre is destroyed the person hears spoken words, but they convey to him no meaning. All that is said to him sounds as if it were in a foreign language: he hears, but he does not understand. This condition is known as *word deafness*, or auditory aphasia. Varieties of auditory aphasia occur. In some cases for example, a patient cannot name objects seen, felt, heard or touched (anomia).

In communicating our ideas, we speak, write, and make gestures. In speaking we make use of the organs of articulation, and this use involves the fine adjustment of a delicate muscular apparatus. In the act of expressing ideas we have to bring into play the memories of the past muscular movements of this articulatory mechanism. These movements were learned by a slow and painful process during infancy. After the power of speech is acquired, the mechanism works readily and almost automatically, because we only have to send a stimulus to the centre which presides over the stored-up memories of the impulses to innervate properly the mechanism of speech. There is, therefore, a centre for the memories of the movements of articulation—a centre which is, of course, closely connected with the motor areas that directly innervate the larynx, pharynx, and oral and facial muscles. When a lesion destroys this centre for speech memories, a person is unable to reproduce the words necessary for expressing an idea; for example, he sees a knife, he knows what it is, but the memory of the motions necessary to express the word "knife" is gone. To him it seems that the name is gone, and that is the common way of expressing it. He cannot say the word "knife." The patient may wish to express the idea of pain. He feels the pain, he knows that he has pain, but he cannot revive those motor memories which are concerned in expressing the word "pain;" he cannot tell, therefore, in words what is the matter with him. When a person is thus troubled, he is said to have a form of motor aphasia for which the particular name given is *aphemia*.

In the same way there is a centre for the memories of the muscular movements concerned in writing; and when a lesion destroys this centre the patient is unable to write, though he may be able to speak. This condition is called *agraphia*.

There is a centre, less well defined, for the memories of the movements used in gesture language, and when this is destroyed the person

is unable to express his ideas by gesture or sign language. This condition is known as *amimia*. A loss of memory of musical expression is called *amusia*.

In some cases, patients are able to speak and write, but they skip words, repeat often, and talk confusedly. There is here a lesion of the tracts associating the language centres, and the condition is called *conduction aphasia*, while to his stumbling speech the term *paraphasia* is given.

Thus symptomatically and psychologically we have the following forms of aphasia:

Sensory.	Alexia.
	Sign blindness.
	Apraxia.
	Word deafness.
Motor.	Aphemia.
	Agraphia.
	Amimia.
	Amusia.
Mixed Forms.	Conduction aphasia.
	Paraphasia.
	Jargon aphasia.

In the examination of a case of aphasia, questions to bring out the following points should be put to the patient:

To test his power of expression:

1. Can he speak voluntarily or use ejaculations expressive of some emotional state?

2. Can he answer questions?

3. Can he count or repeat letters of the alphabet?

4. Can he repeat words? If he can talk, does he talk coherently, or is it a jargon, or does he skip words now and then as he talks?

5. Can he hum tunes?

6. Can he write voluntarily or to dictation? Can he copy?

7. Can he indicate his wants by gestures?

To test his power of understanding language:

8. Does he understand the words spoken to him, or gestures made to him?

9. Can he name things seen, heard, felt or can he name abstract things, like darkness, beauty, sorrow?

10. Can he recall to his mind objects named to him? That is, if the word "poison" is spoken, does he know what it means?

11. Can he read silently or aloud?

12. Does he know the uses of things about him, such as how to use a knife and fork, or a pencil, and what is his general intelligence?

By a series of questions of this sort we can make a diagnosis of the

psychological quality of his speech defect, and say that he has "auditory" aphasia, or "motor" aphasia, or a "visual" aphasia, and so on.

The physician, however, can ignore this kind of diagnosis, to a large extent, and he had best try first to determine the anatomical location of the aphasia, and arteries involved in the lesion. By the method of questioning which has just been laid down, the patient is discovered to have certain peculiar defects of language, and, in accordance with these defects, the physician is able to assign the trouble to one or another portion of the brain.

1. Thus, in one very common form of speech disturbance, we have a fronto-capsular lesion, *i.e.*, a lesion injuring the left third frontal convolution and often extending back so as to involve the internal capsule somewhat. Here, the patient has usually hemiplegia but no sensory disturbance, and his intelligence is quite good. The main characteristic of his speech defect is that of aphemia, that is, a complete inability to say a word, or, at least, only one or two simple words. With this mutism, there is also usually a loss of ejaculatory speech, and of responsive speech, of counting or association speech, and of ability to repeat words that are spoken. Yet, though thus completely aphemic, the *patient can write and read* well, and *understand spoken language perfectly.* The lesion here is in the third left frontal convolution, extending, perhaps, somewhat into the lenticular zone (Fig. 173), and due to involvement of one of the ganglionic arteries, which does not supply much of the corpus striatum. Sometimes there is an involvement of the cortical arteries. Pure frontal lesions are very rare, because the third frontal convolution is not supplied altogether by a single artery but by several. Occasionally this portion of the brain is rather extensively involved, and there may be with some hemiplegia, also evidences of difficulty in understanding spoken language. Sometimes the pure fronto-cortical lesion causes an aphemia, like this described, which is temporary, because the lesion is so small. This last form of aphasia is sometimes known as "pure motor" aphasia or "pure motor subcortical" aphasia.

2. The second type of aphasia is an "occipital" or "parieto-occipital" aphasia, known clinically as "pure alexia."

In this group of cases, the patient can talk well, and understand, and write well, but he cannot read, except a few words or letters, and he cannot copy well. He has generally a hemianopsia, and sometimes the alexia is wrongly attributed to this. Usually there is with it some hemiataxia or hemianæsthesia or hemiparæsthesia, and there may be a very slight temporary hemiplegia. The intelligence of the patient is good. These cases are quite rare also. They are due to a lesion in the occipital lobe, extending sometimes into the angular gyrus. This part of the brain is supplied by the posterior cerebral artery. When lesions of this

artery occur, they involve the visual centre often, and also the so-called association or visuo-psychic centres concerned in reading. These are situated in the angular and in the second occipital gyri. While the lesion is usually due to involvement of the posterior cerebral, alexia may be caused also by lesions of the Sylvian artery, since it supplies some of the angular gyrus, but in this case we get some injury of the temporal cortex, and an alexia which is complicated with other symptoms (Fig. 196.)

3. A third group of aphasias is a "temporal or "parieto-temporal" or the "sensory aphasia of Wernicke." Here the patient is found to have no hemiplegia, or only a temporary and slight one, but his power of coherent speech is seriously injured. He has some voluntary speech,

FIG. 196.—Lesion of Sylvian artery, and softening of temporo-parietal region, with aphasia. (*Spiller.*)

and responsive speech, and may use words very fairly, even loquaciously, but he talks nonsense on account of the confusion, repetition and misplacement of words. He has, in other words, paraphasia or jargon. He cannot name objects shown him, and usually not objects that are heard by him, or felt, or smelt, or tasted. This constitutes a symptom known as "anomia," or "optic aphasia." He can read a little, but very poorly, and write a little and copy, but this is all done very badly. In other words, he has some alexia and agraphia. He cannot understand questions or simple directions, or, if he does understand simple directions, he cannot understand complicated ones, and there is, apparently, some loss of general intelligence. Patients act sometimes rather childishly and emotionally. Perhaps the dominant symptoms may

be said to be this inability to understand, and together with confusion and jargon or paraphasia of speech. With this speech defect there may be, as already stated, a temporary or slight hemiplegia. There is usually some hemianæsthesia or ataxia or astereognosis, or all three of them. There is sometimes a hemianopsia, which may not be permanent. Such persons improve a good deal in their intelligence, but rarely get entirely normal, and they have to be watched rather carefully. They do not show symptoms of any true dementia. The lesion, in these cases, is in the auditory and the auditory psychic area of Campbell, that is to say, in the posterior two-fifths of the first temporal convolution, extending, in severe cases, into the parietal lobe or into the second temporal. There is a cutting off, to some extent, of the association tracts between the auditory psy-

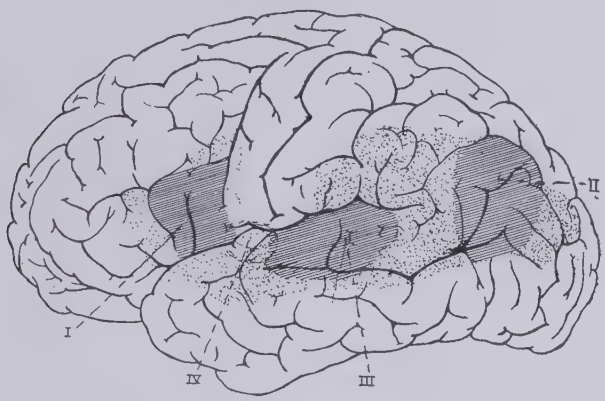


FIG. 197.—Showing the zone of language and the localization of the four types of aphasia. I. Frontocapsular aphasia, characterized by aphemia. II. Parietooccipital aphasia, characterized by alexia. III. Temporal or temperoparietal aphasia, characterized by anomia, some mind deafness, and paraphasia. A small lesion may cause only optic aphasia. IV. Frontolenticular aphasia, the common type of aphasia, characterized by aphemia with much agraphia, alexia, mind deafness, and hemiplegia.

chic and the visual area. The artery here involved is, of course, one of the terminal branches of the Sylvian, or perhaps the trunk of the Sylvian itself. In this latter case, when the lesion is extensive, the speech is apt to be very confused, and almost purely jargon, and there is more likely to be some hemiplegia. Occasionally, a very small lesion of the first temporal alone occurs, and here the patient is simply unable to name objects seen, and often objects that are felt, or heard, or smelt, or tasted. In this case, we have what is called a "pure anomia," or inability to name things. Another term for is is "optic aphasia."

4. The fourth group of aphasias is the "fronto-lenticular" or "lenticular zone" aphasia and "mixed" aphasia. This is the most common of all aphasias. We here find a very extensive aphemia, that is, inability

to talk voluntarily or even to say any words. There is a considerable loss of ability to read and reading is always imperfect; that is, there is some alexia; there is also some difficulty in understanding, at least complicated sentences; that is, there is some mind deafness. There is always a very decided hemiplegia, but rarely any sensory symptom. The lesion here involves the branch of the middle cerebral that supplies the corpus striatum, the internal capsule, and the fibres converging into it from the second and third frontal and precentral convolution. In other words, the lenticular zone is involved and part of the fibres that come from the temporal and frontal areas of speech are destroyed. In some way also, the association tracts from the occipital lobe must be affected, because of the difficulty in reading.

This form of aphasia, which I call, anatomically, the "lenticular zone" aphasia, is called by Marie, an anarthria plus the aphasia of Wernicke. The accompanying diagram will show the anatomical localization of the four different groups of aphasias. And I am sure that the student and physician, in attempting to make a diagnosis of aphasia, will find the task much lightened by turning his attention to the effort to locate, anatomically, the area of the brain and the artery involved.

Conduction and Mixed Aphasia.—There are very few cases of pure conduction aphasia. When it occurs there is paraphasia and paragraphia; the patient repeats words over and over in a kind of verbal intoxication, or mixes things so that the speech is almost gibberish. Still he can express himself and can write, read and understand. The lesion is usually in the island of Reil or the convolutions about the fissure of Sylvius. It is a lenticular zone aphasia.

Practically, conduction aphasia is usually mixed with a visual or auditory aphasia.

Thus we have the following groups of aphasia:

Clinical Type	*Anatomical Seat*
1. Pure motor aphasia.	Fronto-cortical aphasia.
Pure agraphia.	Fronto-cortical aphasia.
2. Pure alexia.	Occipital or parieto-occipital aphasia.
Apraxia.	
3. Auditory aphasia.	
Anomic aphasia.	Temporo-parietal aphasia.
(Sensory aphasia of Wernicke).	
4. Mixed and conduction aphasias.	Lenticular and fronto-lenticular aphasia.

MALFORMATIONS OF THE BRAIN AND ITS ENVELOPES

Congenital malformations of the brain are of little practical importance, for in most cases the monsters cannot live and in all cases they are better dead. I shall simply give a brief enumeration of the important forms.

Abnormalities of the brain.	Anencephaly.
	Micrencephaly and microcephaly.
	Porencephaly.
	Absences or malformations of parts, *e.g.*, cyclopia.
Abnormalities of brain and its envelopes.	Acrania.
	Meningocele.
	Encephalocele.
	Hydrencephalocele.

Anencephaly is always present with acrania. In anencephaly the cerebellum and part of the basal ganglia may be present. In such case the child can live a short time.

Micrencephaly and Microcephaly.—Micrencephaly is a condition in which the brain is only partially developed. If, as is usually the case, the cranium is also abnormally small, it is called microcephaly. It is due, probably, in all cases to an inherent defect in the growth of the brain. Virchow has asserted, however, that there is a form in which the abnormality is caused by a premature growing together of the cranial bones, a micrencephaly being a result of the mechanical condition. An adult cranium whose great circumference measures less than 43 cm. will contain a micrencephalic brain. The normal minimum weight of the adult brain is 960 grams for man and 880 for woman. It should bear the ratio to the body at birth of 14 per cent., and of 2.37 per cent. in adult life (Vierordt).

Porencephaly is often an artificial condition. It will be described later.

In cyclopia there is an undivided anterior cerebral vesicle; the orbits form a continuous cavity with a single rudimentary eye.

Meningocele is a hernia of the brain membranes, arachnoid, and dura mater through a cleft in the skull. In encephalocele the brain also protrudes. Both these forms occur usually in the occipital region and almost invariably in the median line. In hydrencephalocele there is a sac with fluid contents.

DISEASES OF THE MEMBRANES OF THE BRAIN

The diseases to be considered under this head are anæmia and hyperæmia, inflammation of the dura mater or pachymeningitis and inflammation of the pia mater or leptomeningitis.

Anæmia and Hyperæmia of the Membranes of the Brain.—Anæmia of the membranes of the brain is a condition that cannot be separated from anæmia of the brain substance, and will be considered in connection with it. Hyperæmia of the brain membrane, so far as it relates to hyperæmia of the pia mater, must also be considered in connection with hyperæmia of the brain tissue. Dural hyperæmia, or congestion of the dura mater, is a condition which occurs as the result of injuries, sunstroke, and of certain infective poisons, especially that of syphilis. The symptoms are those of pachymeningitis of the slight grade, and will be described under that head. They consist mainly of pain, occasional attacks of vertigo, and sensations of fullness about the head. The treatment is that for the beginning stages of a meningitis.

Inflammation of the Dura Mater or Pachymeningitis Externa.—It has been the custom to describe two forms of pachymeningitis, the ex-

ternal and the internal. Internal pachymeningitis, or hæmatoma of the dura mater, is properly a hemorrhagic disorder, and is described under the head of Dural Hemorrhages. A true inflammation confined to the internal surface of the dura alone is of extremely rare occurrence.

Pachymeningitis externa is a disease that involves, at first at least, the outer surface of the dura, and is usually of surgical origin and interest.

Etiology.—Accidents, injuries, osteitis, caries of the petrous bone in mastoid disease, of the ethmoid bone in ozæna, necrosis, syphilis and erysipelas are the usual causes.

The *symptoms* are local headache, fever, delirium, sometimes even convulsions and paralysis. In the severe cases the disease has usually extended and involved the pia. Pus is generally formed, and burrows between the bone and dura. The disease is recognized mainly by the discovery of the local cause. The *course* is acute or subacute. The *treatment* is a surgical one.

Inflammations of the Pia Mater.—*Cerebrospinal Meningitis.*—Inflammation of the pia mater has the following types: (1) Acute purulent meningitis due to various forms of pyogenic organisms other than the meningococcus; (2) epidemic cerebrospinal meningitis due to a specific meningococcus infection; (3) tuberculous meningitis; (4) serous meningitis and (5) syphilitic meningitis.

Most of all these forms of meningitis may be either acute or chronic the chronic form being usually simply a sequela of the acute.

ACUTE PURULENT CEREBROSPINAL MENINGITIS

Etiology.—Acute leptomeningitis is due to an infective process.

The infecting organism is usually a streptococcus, pneumococcus, or staphylococcus. The meningitis caused by the meningococcus is of a special type described later. The streptococcus is the most frequent infecting organism. The infection is almost always secondary to a focal lesion or general infection.

The commonest focal lesions are disease of the ear, and of the nose and accessory sinuses. Fractures of the skull and other wounds of the head, or spine may become infected and lead to meningitis. Over-doses of salvarsan introduced into the spinal canal may cause by chemical irritation a meningitis. Brain abscess, and infected sinus thrombosis may be causes.

Just now it is stated that purulent meningitis is seen most often by obstetricians from sepsis and by otologists and rhinologists.

Pneumonia is the most frequent infective disease in which the pyogenic organisms are carried by the blood. After this come erysipelas, sepsis, scarlet fever, more rarely endocarditis, empyema, rheumatism, measles, typhoid fever and mumps. Alcoholism and a generally lowered state of vitality from malnutrition predispose to the infection. Insolation can

of itself not cause it. The disease is more frequent in males, and is distributed through all ages of life, though it occurs oftener in the young.

Symptoms.—The symptoms in the various types differ somewhat, but have a general similarity. They are to be broadly grouped into the prodromal, the irritative, the depressive, and the paralytic stages.

Prodromal symptoms are shorter and less marked in simple meningitis than in tubercular. They are really the symptoms of a general infection. The patient suffers from malaise, languor, headache, vertigo, irritability, loss of appetite and vomiting. Of these symptoms headache is the most notable.

[The description of the symptoms of epidemic cerebrospinal meningitis given later answers for the common purulent form, except for certain variant types.]

Course and Duration.—The disease may begin suddenly, and the patient pass at once into the comatose state, dying in a few days. Usually the process lasts one or two weeks; it may be prolonged for several weeks.

The **prognosis** is very grave, but it is less serious than in tuberculous meningitis and more serious than in the cerebrospinal form.

The **diagnosis** is based on the presence of an exciting cause, such as disease of the ear or nose, trauma, infective fevers, and upon the presence of the symptoms given. It is usually easily recognized, the main difficulty being to distinguish it from other forms of meningitis.

To determine this, it is advisable to make a lumbar puncture, draw off the fluid, and examine it for bacteria and the presence of cellular elements. (See Epidemic Cerebrospinal Meningitis.)

Pathology.—The disease is a fibro-purulent or purulent inflammation. It involves usually the base more than the convexity, but the reverse may happen. The ventricles are often involved and may be independently inflamed. There are descriptions, therefore, of simple basilar meningitis, meningitis of the convexity, and ventricular meningitis or ependymitis. The inflammatory deposits are most conspicuous along the course of the Sylvian fissure and the vessels branching from it, about the optic chiasm, and at the posterior and under surface of the cerebellum and the sides of the pons. It may lie only in the subarachnoid cavity, but usually the arachnoid and sometimes the dura are implicated. There is increase of fluid in the ventricles and arachnoid cavities, and this fluid may be turbid. The surface of the ventricles may show an inflammatory process

The micro-organisms found in meningitis are as already stated the pneumococcus, the streptococcus, the staphyloccus and a bacillus resembling that of typhoid fever. Still others have been described, and the process may be a mixed infection.

Treatment.—Prophylaxis is the most important measure, as there is no specific treatment. Chronic disease of the ear and nasal sinuses should

be attended to, and injuries of the skull treated with the strictest regard to antisepsis. When the disease is developed, the indications are to treat the original focus of infection (ear, etc.) to open the skull and decompress and drain in certain cases; to combat the general sepsis. The treatment is therefore often a surgical one. To combat the infection there are immunizing sera from which not much has been accomplished. Flushing the blood with intravenous injections of saline tranfusions, epidermoclysis and proctoclysis may be tried. Urotropin and formaldehyde have not proved useful. Recently the use of watery solutions of sulphate of magnesium have been strongly recommended.

EPIDEMIC CEREBROSPINAL MENINGITIS

(Spotted Fever)

Cerebrospinal meningitis occurs as an epidemic and endemic disease caused by the diplococcus intracellularis meningitis.

It is a disease which has invaded practically all of Europe and has occurred in the East and in Africa. The most extensive epidemics have occurred in the United States, affecting all the states and spreading to Canada, but not to the countries in the South. The disease occurs sporadically in the large cities of the United States during the winter. The epidemic occurs periodically at irregular times. The disease occurs at all ages but children are the most affected.

In New York City about two-thirds of the cases were in children under ten years of age, but about 16 per cent. occurred in persons over twenty and it may attack persons as old as fifty. The sexes are about equally affected. Race and nationality seem to have no particular influence although in the South the negroes are less affected than the whites.

The disease occurs in the winter months, beginning sometimes in late October and ending in May but sometimes not beginning until the first month of the spring. Unusually cold weather seems to be associated with the height of the epidemic.

The disease is most prevalent among poor and in over-crowded dwellings. In New York three-fourths of the patients lived in tenement houses.

The disease is very slightly infectious. Fatigue and ill health predispose somewhat to its development. The infection occurs through the nose and throat, and nasopharyngitis predisposes to the entrance of the organism. The organism is carried by healthy individuals and the disease is propagated to a great extent through these carriers. It has been estimated that during an epidemic there are ten to thirty healthy carriers in proportion to each single person who actually has the disease. The nasal and oral secretions of the healthy carriers give positive cultures of meningococci. Usually the organism spontaneously disappears

from the nasopharynx of the carrier in a few days, but there are instances in which a healthy person may harbor the coccus for one or more months.

The invasion of the body by the meningococcus is probably accomplished by the passage of the organisms from the nasopharynx into the blood stream, resulting in a primary bacteræmia, and later the bacteria become localized in the meninges. It is possible that the meningococcus passes directly into the cranial cavity from the nose and throat by means of the lymphatics, but the weight of evidence is against the view.

Pathology.—An exudation of serum, fibrin, and pus covers the pia of the brain and cord and infiltrates its meshes. There is some involvement of the superficial layers of the cortex in the inflammatory process. The blood-vessels of the meninges are engorged and surrounded by a cell infiltration. The inflammation extends along the sheaths of the cranial nerves and also into the ventricles. The ventricles are distended with a mixture of cerebrospinal fluid and exudate, and their walls are red and oedematous. The cerebrospinal fluid is increased in quantity. In chronic cases the pia arachnoid may show some cicatricial thickening.

The organism, a Gram-negative, biscuit-shaped diplococcus, is to be found in the exudate and in the cerebrospinal fluid, where it is seen in the polynuclear leukocytes and also free. It may be obtained by culture from the nasopharynx and from the blood.

Symptoms.—The disease begins as a constitutional infection produced by the passage of the organism into the general system and this is followed by local symptoms produced by the localization of the organism in the meninges. The period of incubation—*i.e.*, the time between the invasion and the development of the constitutional symptoms, probably ranges between one and five days.

The constitutional symptoms corresponding to the period of simple bacteræmia are very much like those of influenza. The disease comes on acutely with chilly feelings or a severe chill. There is fever, throbbing headache, restlessness, irritability, and vomiting. The mind is clear but the patient is over-sensitive. Often the pupils are dilated and sluggish, and there may be photophobia. A little early stiffness of the neck and tenderness at the angle of the jaw may be present. Herpes and a petechial eruption are occasionally seen. Sometimes there is irregularity in breathing, particularly while the patient is asleep. Deep and uncontrollable sighing is quite common. The temperature varies; sometimes it is very high, and sometimes low. In young children there may be a little bulging of the fontanelles, and in adults Macewen's sign can often be obtained. It consists in a dull tympanitic note brought out by percussing with the finger over the fronto-parietal region of the skull. It is most easily obtained if the patient's head is lifted a little and bent

slightly toward the side to be examined. The sign indicates an excessive amount of fluid in the ventricles. Blood examination at this stage usually shows a moderate leukocytosis with relative polynucleosis. Lumbar puncture reveals an increase of pressure. The fluid is usually perfectly clear, however, although a few free meningococci are sometimes to be found. These symptoms usually last for twenty-four to thirty-six hours, when the meningeal symptoms make their appearance. In some cases the pre-meningitic symptoms are entirely absent, and in others a history of them is obtained only by careful questioning of the family.

The stage of active meningitis begins with an increase of all the foregoing symptoms. The headache becomes terrific and the patient complains of it bitterly. A number of general convulsions may occur, particularly in children. Sometimes consciousness is suddenly lost. There is extreme restlessness and frequently delirium. Vomiting is frequent and severe, and in many cases uncontrollable. Usually it is independent of nausea, and it may be projectile. The patient suffers severe pain in the back of the neck, along the spine, and in the legs. The neck becomes stiff, the head may be retracted, and in some cases there is rigidity of the spine or opisthotonos. Photophobia is often very marked. The patient is greatly prostrated. Active delirium and restlessness alternate with deep stupor, in which, however, the patient usually shows some response if he is disturbed. There may, however, be periods in which the mind is clear. The patient's face bears an anxious expression and often is cyanosed. The rigidity of the neck may be so great that with a hand placed under the patients head one can lift his body from the bed without there being any appreciable bending of the neck. An attempt to move the neck in any direction is very painful, and there is tenderness to pressure on the posterior neck muscles. The pupils are generally dilated, and react to light sluggishly or not at all. Often there is some strabismus, which may be transitory. Other cranial nerve palsies may occur, and there may be clonic or tonic spasms of the extremities. In young children there is pronounced bulging of the fontanelles. *Tache cerebral* is readily obtained. Kernig's sign is early and constant (Figs. 195, 198). This test is made by flexing the patient's thigh to a right angle on the abdomen and then attempting to extend the leg on the thigh. This attempt, in meningitis cases, is resisted by a sudden spasm of the hamstring muscles and the patient cries out in pain. The abdominal reflexes are often missing. The tendon jerks frequently are absent, but they may be exaggerated. Rarely there is a dorsal flexor response of the toe. Brudzinski's signs of meningitis may be present. In the first sign, if one of the patient's thighs is flexed upon the abdomen as far as possible (the leg being flexed on the thigh), the patient then draws up the other into a similar position. In the second, with one leg and thigh extended and

the other flexed as before, the flexed limb is lowered to the extended position by the examiner, whereupon the patient draws up the other limb to a position of flexion on the abdomen. The "neck sign" of De Lepinay can usually be obtained. It consists of a flexion of the knees and thighs which occurs when one attempts to bend the patient's neck forward.

Most cases of meningitis show some respiratory irregularity. Connor and Stilmann found Cheyne-Stokes breathing in 53 per cent. of their cases and Biot's breathing in 27 per cent. In only 5 per cent. of all their cases did they fail to find some sort of irregularity of breathing. Biot's breathing ("cerebral breathing") is thought to be typical of meningitis as distinguished from other cerebral conditions causing disturbances of breathing. It is characterized by periods of apnœa of varying length and occurring at irregular intervals (as distinguished from the rhythmical

Fig. 198.—The Kernig sign.

periods of apnœa in Cheyne-Stokes breathing which alternate with periods of gradually increasing and gradually decreasing respiratory movement), constant irregularity in the rhythm and the force of individual respirations, and the frequent occurrence of deep sighing respirations.

The temperature in meningitis is usually very irregular. It may be extremely high (106° F.), or it may be little above normal. A septic temperature may occur, or the fever may be high continuously. The pulse rate is also irregular. It may be very slow—50 or less—but more often it is rapid. The pulse may be intermittent or show frequent changes in rate.

Generalized skin eruptions occur in only a small percentage of cases. Petechiæ if present are usually profuse, and are distributed all over the trunk and extremities. Purpuric eruptions do occur, but are less common. Herpes, however, is a skin condition very commonly found.

Lumbar puncture shows that the intra-spinal pressure is abnormally increased. The fluid is turbid, globulin and albumin tests are strongly positive, and cells are numerous, most of them being polynuclears—100 per cent. in some cases. Stained smears from the sediment obtained by centrifuging show the meningococcus—in varying numbers—*i.e.*, Gram-negative, biscuit-shaped diplococci, both intra- and extra-cellular. Cultures show a growth of the meningococcus after eighteen to twenty-four hours' incubation. Elser and Huntoon were able to make a positive diagnosis from the examination of the spinal fluid in 92.5 per cent. of over 200 cases examined. No altogether satisfactory serological tests for meningitis have yet been devised.

Blood examination shows a leukocytosis of between 20,000 and 45,000. The polynuclears are markedly increased, there is a relative decrease in the large mononuclears, and in very young children a lymphocytosis had been observed in rare instances. The meningococcus can be obtained from the blood by culture in a certain number of cases. Elser had positive results in 26.5 per cent. of the cases examined.

Course and Prognosis.—The disease may run a short and malignant course killing the person in a few hours or one or two days. In moderate cases it lasts two or three weeks. A large number of different varieties of the disease have been described, such as the abortive form, the fulminating form, and the typhoid form. Affections of the joints are the most frequent complications of the disease. There may also occur septic pneumonia, pyelitis, ulcerative endocarditis, pericarditis, neuritis, and phlebitis. In some cases the disease leaves a chronic hydrocephalus, and there may be some permanent mental impairment. Permanent paralysis of some of the cranial nerves, or, much more rarely, of one or more of the extremities may occur. Deafness is quite common, and blindness and other eye troubles sometimes are observed.

A certain type of case lapses into a state of chronic meningitis, which runs a course of a few weeks' to two or three months' duration, and usually terminates fatally. It is characterized by extreme emaciation and severe cerebral pressure symptoms. The temperature is extremely irregular. It may be normal or subnormal, or, after having been low for a considerable time, suddenly rises and remain high and irregular. Chills may occur irregularly. The emaciation is most remarkable. Though many of these patients take large quantities of food and appear to digest it properly, they become rapidly reduced to skeletons, the like of which is hardly to be seen in any other disease. The patient is usually drowsy and stupid but there may be active delirium. Complete coma occurs in the final stages. The patient's eyes are wide open and the face expressionless. Spasms and convulsions are common. Vomiting is frequent and the bowels are constipated. Macewen's sign or bulging of

the fontanelles may be present. Rigidity of the neck and Kernig's sign usually continue present, but not always so. The respirations are slow and irregular, and the pulse rapid, weak, and intermittent. Strabismus or some cranial nerve paralysis is usually present. The tendon jerks are absent or diminished. Incontinence of urine and fæces is practically invariable. Bed-sores develop readily.

Another form of chronic meningitis, the so-called posterior basic meningitis is distinguished by the fact that there occurs a shutting off of the communication between the ventricles and the sub-arachnoid space. The inflammatory process thus becomes encapsulated in the ventricles, and whatever inflammation was going on in the subarachnoid space usually subsides so that the fluid obtained by lumbar puncture may be sterile. More commonly, no fluid at all is to be obtained.

Fig. 199.—Cerebrospinal meningitis, terminal stage.

The condition of posterior basic meningitis may develop at the outset of the infection, but more commonly it appears in the course of a subacute or ordinary chronic meningitis.

The symptoms are those of a rather mild sepsis with marked cerebral pressure. The rigidity of the neck and retraction of the head are extreme, and a high degree of opisthotonos is the rule. The patient is apathetic and stuporous, and convulsions are very common. Usually the patient lies motionless with the extremities rigid in tonic spasm. The "hydrocephalic cry" which is altered at intervals without relation to pain or other stimulation is a common symptom. The eyes are wide open, the lids retracted, the pupils do not react to light, and there may be some exophthalmos. Various palsies are present. The patient may remain in this condition for several weeks. There is practically always a fatal termination.

The prognosis of the usual form of meningitis is always grave. The

mortality in different epidemics has varied from 20 to 80 per cent. Only a small percentage of those cases that become chronic recover. Sophian thinks that the development of Biot's breathing in either acute or chronic cases is of prognostic value indicating almost without exception a fatal termination.

Diagnosis.—The differential diagnosis of epidemic cerebrospinal meningitis is to be made from tuberculous meningitis, from meningitis caused by strepto- and staphylococci, the pneumococcus, and other pyogenic organisms; from poliomyelitis with meningeal symptoms, and from the so-called meningismus which occurs in various infectious diseases.

Meningitis due to streptococci, pneumococci, and similar organisms presents the same clinical picture that is found in epidemic meningitis. The history of an epidemic, on the one hand, or of some preceding focus of infection, as for instance otitis media or broncho-pneumonia on the other, may help toward making a diagnosis. Ordinarily, a positive differential diagnosis can be made only through the results of the examination of the cerebrospinal fluid.

In tuberculous meningitis as a rule the children are younger—two years or less of age. A family history of tuberculosis may be obtained, or some family form of tuberculosis can be made out. The child is apathetic and stuporous instead of irritable and restless. Cranial nerve palsies occur early. Rigidity of the neck and Kernig's sign are usually moderate and the temperature at first is not very high—101 to 102. There is no eruption. The course is acute and death occurs in two to four weeks after the beginning of the disease. Lumbar puncture gives an abundant fluid, which usually is clear with white specks of fibrin floating in it. If the fluid is allowed to stand for a time a network of fibrin forms. There is a lymphocytosis instead of a polynucleosis. Careful search may reveal the presence of the tubercle bacilli, and cultures fail to show the diplococcus intracellularis or other pyogenic organisms. In the cerebral type of poliomyelitis the patient is apathetic and stuporous, but he can be roused and when this is done his mind is quite clear and he can answer questions intelligently. Though there is restlessness and irritability these symptoms are much less marked than in meningitis. Rigidity of the neck and Kernig's sign, if present, are not very marked. The profound disturbances of respiration and pulse which occur in meningitis are absent in poliomyelitis. The paralyses in poliomyelitis occur early and are comparatively extensive, while in meningitis the palsies occur later and ordinarily are less extensive. Usually in poliomyelitis the constitutional and the cerebral symptoms rapidly improve as soon as the paralyses occur, while in meningitis the occurrence of the palsies is ordinarily attended by an exacerbation of the constitutional and cerebral symptoms. The fact that poliomyelitis is a summer

disease and meningitis a winter one should not be forgotten in making a diagnosis.

The fluid obtained by lumbar puncture in poliomyelitis is clear with white flecks floating in it, and, in fact, appears altogether like that obtained in tuberculous meningitis. There is, however, less tendency for it to form a fibrin network on standing, and, if a network does form, it is less dense than in the tubercular condition. The fluid is usually less abundant and under less pressure. Globulin tests are positive, but less strongly so than in meningitis. The cells are increased but not to the extent to be found in meningitis and there is usually a lymphocytosis. In short the fluid findings of poliomyelitis are much more likely to be confused with those of tuberculous meningitis than of the epidemic variety. Bacteriological examinations of the fluid are negative for tubercle bacilli and other organisms, and the inoculation of guinea-pigs as a test for tuberculosis is negative.

The diagnosis of meningitis from meningismus is usually not difficult. The presence of the primary disorder causing the meningismus can usually be made out. Unless there is delirium the patient is clear and intelligent and, though irritable, does not show the same crying and complaining that occurs in meningitis. Kernig's sign, bulging of the fontanelles, and Macewen's sign are moderate or absent. Lumbar puncture settles the question if the diagnosis cannot be made without it.

Treatment.—*Prophylaxis.*—Cases of epidemic meningitis should be quarantined until cultures from the nasal and pharyngeal secretions are negative for the meningococcus. Healthy people who come in contact with these patients should be examined in the same way, and if their cultures are positive they should be quarantined also, and directed to use a disinfecting spray of hydrogen peroxide, preceded by salt solution, until negative cultures are obtained, thus doing away with the danger of their infecting others or of becoming infected themselves.

The general treatment is directed toward supporting the patient and combating the toxæmia. Sustaining food should be given and such stimulants as digitalis when indicated. Bromides, chloral and morphine are used as sedatives. Ice bags to the head and cool bathing for high temperatures are also employed. Lumbar puncture is employed for the purpose of diminishing pressure by draining off the fluid.

The specific treatment consists in the administration of the Flexner serum. A lumbar puncture is done and the cerebrospinal fluid allowed to flow until it comes from the needle at the rate of one drop every three to five seconds, which indicates roughly that about the normal degree of pressure has been attained. Then from 5 to 40 cc. of the Flexner serum, depending on the age of the patient, is slowly and carefully injected into the spinal canal through the puncture needle. The safest

method of injecting the serum is that of allowing it to flow into the spinal canal by gravity, using instead of a syringe a funnel attached to a twelve inch tube, and watching the blood pressure meanwhile as an index of when the flow should be stopped. A fall in blood pressure of 10 mm. of mercury indicates that enough serum has been injected (Sophian). The injections are repeated every day for three or four days and then less often. The best guide to the proper frequency of the injections is the examination of stained smears from the sediment from the fluid. As the patient responds to treatment the number of the cocci decreases and those that are found are intra-cellular. Numerous and extra-cellular organisms indicate that an injection is required on the following day. The use of this serum has given very satisfactory results. In different epidemics where a mortality of from 50 to 90 per cent. occurred in those cases not treated with serum, the mortality was reduced to between 13 and 50 per cent. by its use. H. W. Frink, M. D.

TUBERCULOUS MENINGITIS

This is a form of meningitis due to infection with the bacillus tuberculosis. It differs pathologically from other forms in the character of the infective organism; anatomically, in the fact that the inflammation is usually and chiefly basilar and never purely purulent; etiologically, in that it chiefly affects young children; and symptomatologically, in the presence of prodromata and a more irregular course.

Etiology.—Tuberculous meningitis occurs chiefly between the ages of two and ten, oftenest between two and five years, sometimes in infancy, rarely in adult life, very rarely after the age of fifty. Males are rather more subject to it. A hereditary history of phthisis, bad hygienic surroundings, and the presence of tuberculosis elsewhere in the body predispose to it. Tuberculous milk is probably one source of the infection. The eruptive fevers, especially measles, blows on the head, and great emotional excitement appear to act as exciting causes.

Symptoms.—The child in the prodromal stage becomes peevish, irritable, loses interest in its play, the appetite is gone, and there may be explosive vomiting. Sleep is restless and disturbed. Paroxysms of severe headache occur. After two or three weeks, or sooner, these symptoms increase; the child becomes dull, moody, weak, and takes to his bed. He lies there with clouded consciousness, dozing at intervals; often the cheeks are ruddier than usual. Occasionally he utters a piercing cry as if in great pain. He is easily disturbed by noises, and shows himself generally hyperæsthetic. He sighs deeply, picks his lips or the bedclothes. The neck is now felt to be rather stiff; the reflexes are increased; and the abdomen retracted or perhaps distended. The temperature runs an irregular course but is not high. The pulse is slowed

and irregular, sometimes becoming very fast on slight exertion. The respiration is disturbed and sighing. The pupils are often uneven, usually contracted, and do not react well to light. The patient finally passes into deep coma, with rise of temperature. The picture is now one of the last stages of meningitis, and death occurs in about three weeks. The *special symptoms* and *signs* of meningitis of other types are present in this later stage. The condition of the cerebrospinal fluid is described elsewhere. (See Epidemic Cerebrospinal Meningitis).

Pathological Anatomy.—In rapidly fatal cases, with severe symptoms, there may be only an intense congestion of the brain with numerous miliary tubercles in the pia mater at the base and over the convexity. Here we must assume that a bacillary toxin causes the symptoms. In most cases there are decided deposits of tubercles at the base, with fibrinous inflammatory deposits about the optic chiasm, along the fissure of Sylvius, at the sides of the pons, and elsewhere. Miliary tubercles are seen scattered over the convexity and in the choroid and ventricles. They are generally found in the spinal membranes also, especially over the cauda equina. The tubercles lie beneath the pia surrounding the small vessels. They may coalesce into large tuberculous nodules. There is usually an increase in the arachnoid fluid, and in most cases an increase in the ventricular fluid. Somewhat rarely there are very great distention of the ventricles and compression of the convolutions. Small spots of softening may be seen from obliteration of the vessels by the tubercles. The bacillus tuberculosis is found in the tuberculous nodules.

Diagnosis.—As regards the form of the disease, this is based on the hereditary history, the age, the existence of tuberculosis of the lungs or other organs, and the peculiar prodromata of the disease. Occasionally tubercles can be seen on the choroid. Lumbar puncture of the spinal canal with withdrawal of fluid and its examination furnishes the most certain method of diagnosis. (See diagnosis of meningitis.)

Prognosis.—This is usually absolutely bad, yet post-mortem observation of patients dying with practically no inflammatory change makes it seem possible that the disease might be checked, and a good many cases are reported in which this apparently has been done. Some of these are, however, probably cases of hereditary syphilis.

Treatment.—So far as is now known, this is not different from that given under the head of meningitis elsewhere.

HYDROCEPHALUS

Hydrocephalus is a condition in which there is an excessive amount of fluid in the ventricles and subarachnoid or subdural cavity of the brain. When this fluid is in the ventricles it is called internal hydrocephalus, and practically most hydrocephalus is internal. In external

hydrocephalus the fluid is in the subarachnoid or subdural spaces. External and internal hydrocephalus may coexist. Hydrocephalus is a symptom of several morbid conditions, but in general we may divide it into the chronic primary and secondary symptomatic forms.

Chronic Primary Hydrocephalus.—This is a disease of infancy, characterized by a gradual enlargement of the head, with mental deficiency and symptoms of brain irritation caused by an accumulation of fluid in the ventricles of the brain.

Chronic hydrocephalus is not an inflammatory process, but one due to mechanical causes or to defects in structure or development. The fluid always accumulates in the ventricles of the brain; hence chronic hydrocephalus is always *internal*. The so-called external forms of hydrocephalus are inflammatory or else are secondary to meningeal hemorrhage or brain atrophy.

Etiology.—Four out of five cases begin at birth or within the first six months of life. Congenital anomaly, intra-uterine meningitis, *syphilis*,

Fig. 200.—Chronic hydrocephalus.

alcoholism, lead poisoning in the parents, and some unknown family taint predispose to the disease. Malnutrition and rickets are also factors.

Symptoms.—The head may be so large at birth that instrumental help is needed. More often the parents notice a gradual increase in the size of the child's head, beginning soon after birth. The forehead bulges, the occiput stands out, the fontanelles and sutures widen, and pressure shows evidence of fluctuation. Meanwhile the face does not grow much and the result is to give triangular shape to the head. It may measure twenty-four, twenty-seven and one-half (Minot), thirty-two (Bright), and even forty-three inches (Klein) in diameter. These extreme measurements are reached only after one or two years. With this abnormal growth of the head, mental and physical symptoms appear. The infant is restless and irritable; its appetite may be good, but the general nutrition is poor and its bodily growth is retarded. The mind does not develop; usually it does not or cannot learn to walk. It may be unable

to support the weight of its head. There is strabismus and sometimes optic atrophy. The pressure of the dropsy thins the orbital bones and forces down the axis of the eyeballs (see Fig. 200). Vomiting, coma, and convulsions eventually appear, and the child usually dies of exhaustion or some intercurrent disease in two or three years.

In some cases the trouble is less serious, it ceases to progress, the bones solidify, and the child grows up with good intelligence.

The hydrocephalus which is associated with the brain atrophy of insanity and old age (hydrocephalus ex vacuo) or with general dropsical conditions has no kinship with the process we are now describing.

Pathology.—The disease is due to the gradual accumulation of a serous fluid in the ventricles of the brain. The cause of this is an inflammatory or developmental obliteration of the aqueduct of Sylvius or the foramen of Magendie and the adjacent lateral foramina of Luschka. This prevents the escape of the ventricular fluid into the subarachnoid cavity. Contributing factors are congenital or acquired defect in the villous processes of the pia arachnoid, by which the fluid normally is able to pass into the venous circulation; and a rachitic and easily yielding skull. Hydrocephalus is not, therefore, an inflammatory but a mechanical process. The lateral ventricles when the cerebrospinal fluid is secreted are principally and often solely affected, and these are so distended as to press out their cerebral walls, flattening the convolutions and turning them into a thin shell often less than a quarter of an inch in thickness. Sometimes only one lateral ventricle, and in rare cases only the fourth ventricle, is affected by the dropsy.

The diagnosis has to be made from rickets and an acute inflammatory process. In rickets the head is square, the fontanelle does not bulge, the enlargement is less, and there are signs of the disease in the bones elsewhere.

Prognosis.—The congenital cases usually result fatally in a few months, or at least before the third year. Those developing in infancy may persist for four to six years; and in mild cases the disease ceases to progress and a fairly healthy adult life is reached.

Treatment.—A great many measures have been recommended, but there is no unanimity about them. The inunction of mercury and the administration of iodide of potassium combined with intravenous injections of salvarsan are indicated if tests show the presence of syphilis.

Tapping the ventricles in various ways has been recommended, but I have seen no good results. Tapping the spinal cord every three days and later every week is said to be of some service.

Secondary or acquired hydrocephalus is usually caused by an attack of acute meningitis, or by tumors. It may also be due to ependymal inflammation and to obstruction of the veins of Galen by thrombosis or

other mechanical cause. In acute meningitis this is a frequent condition and cause of death. In other cases infants survive the meningitis and, with a growing head, develop symptoms later of the hydrocephalus. In the acute conditions the symptoms may run a varying course owing to the partial and irregular escape of the fluid. The patient, if the disease progresses, becomes stupid and comatose and develops paraplegia and rigidity of the body, retraction of the head and trunk, and great emaciation. The treatment is the same as that for the primary form and consists in puncture of the ventricle or corpus callosum and withdrawal of fluid or a decompressive operation (Cushing). The results of surgical interference have so far not been satisfactory.

ALCOHOLIC "MENINGITIS" (SEROUS MENINGITIS, "WET-BRAIN")

Alcoholic meningitis is a clinical term used to indicate the peculiar group of cerebral symptoms which is seen in persons who have succumbed to the effects of prolonged alcoholic intoxication. The disease is not a true meningitis but an acute toxæmia of the brain with serous effusion; but it may be called, for the purpose of convenience, an alcoholic serous meningitis. It should not be confounded with the acute serous meningitis following infections and trauma.

Etiology.—The disease occurs oftenest in men simply because of the more frequent indulgence of the male sex in alcohol. It rarely develops until a person has been drinking eight or ten years, and, consequently, affects people oftenest between the ages of thirty and forty. The exciting cause is commonly alcohol and in this country whiskey or what are known as "hard drinks," but beer and ale will accomplish the same result. I have rarely seen the disease in wine drinkers. The persistent use of morphine, cocaine, and chloral may lead to much the same condition. The exciting cause is usually a continuous drinking bout of two or three weeks, ending in delirium tremens. The delirium tremens, however, is not by any means always present. The patient may pass from a condition of prolonged intoxication into the condition of alcoholic meningitis or "wet-brain."

Symptoms.—In case delirium tremens has occurred, the patient after two or three days of prolonged delirious excitement gradually sinks into a semicoma. This is accompanied by a muttering delirium. The patient is sufficiently conscious to have flitting delusions and hallucinations of sight and hearing. At this time he is able to drink and take food; the pulse is rather rapid, the temperature is usually normal or may be raised one-half or one degree. The skin is hyperæsthetic and pressure upon the muscles of the arms or legs or abdomen causes pain. The pupils are usually rather small. Often at this time conjunctivitis and keratitis may appear. After a few days the patient's stupor becomes deeper and

he can be aroused only with difficulty. The arms and legs are now somewhat stiff, the reflexes are exaggerated, the neck is somewhat stiff and slightly retracted, and attempts to move it cause expressions of pain. The Kernig sign is present. The abdomen is retracted and the skin and muscles are still very hyperæsthetic. The lids are closed. The pupils are small and do not react well to light. The tongue is coated and usually dry, and urine and fæces may be passed involuntarily. The patient may linger this way for several days more. The pulse becomes rapid and feeble, the extremities are stiff and cold. The skin is dry and loses its elasticity, so that when pulled up between the fingers it stays in folds. "Putty skin" is a good name for this. The coma deepens, the temperature may rise to 103° or 104°, and symptoms of pneumonia may appear as the scene closes. On the other hand in some cases the patient does not pass into the worst stage, the mind becomes clearer, the hyperæsthesia lessens, food is taken better, and the bowels are moved voluntarily. Improvement continues and in three or four weeks the convalescence begins.

Pathological Anatomy.—I have made autopsies and careful microscopical examinations in over twenty cases of the character just described. In nearly all the brain is found to be somewhat pale, the arachnoid contains two or three ounces of serous fluid, the subarachnoid space is saturated with fluid, and the ventricles are dilated. Sections through the brain sometimes show punctate hemorrhages and in rare cases spots of hemorrhagic extravasation are seen surrounded by softening. Occasionally the beginning of a suppurative cerebral meningitis will be seen. Microscopic examination shows in the uncomplicated cases that there is no true inflammatory process. There is often congestion, but not always; the commoner condition is an œdema of the brain tissue, the perivascular and pericellular spaces being dilated. The nerve-cells show various stages of degeneration, not pigmentary in character, however. The chromophilic granules are often unstained, or, if stained, have lost their true relations, and seem broken down. The cell outlines are irregular; the nucleus lies near the periphery of the cell, and in some cases has broken out and escaped from it. Sometimes there is a large number of neuroglia cells in the pericellular spaces. The disease is undoubtedly, primarily at least, a toxæmia not due directly to the influence of alcohol but to the poisons which have developed in the body as a result of the condition of inanition and the paralysis of the digestive function caused by the prolonged ingestion of alcohol and abstinence from food. The cell degeneration is more like that which is known as "degeneration from a distance," such as is seen in nerve-cells when the neuraxon is destroyed. It is a degeneration which affects especially the body of the cell and not so much the nucleus; hence the remarkable power of recovery from this condition which so many people show.

The diagnosis of the disease is to be made from ordinary suppurative meningitis, from acute serous meningitis due to infection, and from acute encephalitis. In most cases the history of the patient is quite sufficient to establish the diagnosis. The symptoms of themselves are almost identical with those of ordinary acute suppurative meningitis. The only distinctions which I have been able to observe are that in suppurative meningitis there is more fever, there is less of the low delirium, hallucinations are rare, and there is an earlier and more profound coma. In other words, it is an acuter and more severe malady than alcoholic meningitis. The absence of convulsions and paralysis and the presence of hyperæsthesia, rigidity, and contracted pupils, as well as the absence of pyrexia, are usually sufficient to distinguish the disorder from encephalitis or encephalitis complicated by alcoholic meningitis. Lumbar puncture will settle the diagnosis and often prognosis. The cerebrospinal fluid is usually in excess and under great pressure; it is clear and colorless, unless there has been a superimposed infection as sometimes occurs, when evidence of inflammation will be found.

The prognosis is bad when the disease has become well developed and when decided coma and rigidity have set in. A prognostic criterium which I have long used and which is fairly accurate is this: if the patient has not a very stiff neck and back he will get well.

The treatment of the disorder should be instituted at the very beginning. If there are still any relics of the debauch, as shown in the condition of the stomach or intestinal tract, the stomach should be washed out and, at all events, a thorough purge should be given. The patient should then be fed most liberally with hot milk given every two hours; beef tea and an egg beaten up in milk may also be given, but the condition of practical starvation on the part of the patient should always be borne in mind. Stimulants in the shape of whiskey should not be administered if it is possible to avoid it, but strychnine in doses of one-sixtieth of a grain every two hours is often useful. An ice cap may be applied to the head and at times leeches or large blisters seem to be useful, applied to the back of the neck. The patient, however, should not be much depleted. Large doses of ergot have been recommended. When he becomes comatose it means that the ventricles and arachnoid cavities are becoming filled with water. At this time tapping the spinal cord may be tried. I have done this in a number of cases and have at times removed two or three ounces of fluid with some amelioration of the symptoms and never any bad results, but I have never seen it do any permanent good.

CEREBRAL HYPERÆMIA AND ANÆMIA

Active Cerebral Hyperæmia.—The circulation of the blood in the brain is regulated mainly by the vasomotor splanchnic nerves, a rather

clumsy system by which a mass of blood is thrown into the abdominal cavity, or pressed out of it, thereby decreasing or increasing the cerebral flow. In addition to this, the cerebral blood-vessels have a nervous supply which probably controls the finer and the more local variations in calibre.

The total amount of blood in the cranial cavity is always about the same. There can be no cerebral hyperæmia, therefore, characterized by a large increase in the intra-cranial content of blood. But the flow of blood through the brain may be very much increased in rapidity; so that the amount of blood flowing through the brain in a given time is greater than the normal, or average.

This physiological cerebral hyperæmia accompanies conditions of over-activity and over-stimulation of the brain, and is associated with increase in mental activity; but cerebral hyperæmia is here a consequence or associated phenomenon rather than the cause, and there is no pathological condition which can be, strictly speaking, called "cerebral hyperæmia."

Passive or venous cerebral hyperæmia occurs when there is some blocking of the venous current and damming back of the blood. Here the venous stasis causes an increase in intra-cranial pressure and an anæmia of the capillaries. The blood current is slowed-up and in addition to that, the proportion of venous blood to arterial is increased. The blood supply is not of as good quality as normal, hence, in passive hyperæmia of the brain there may be symptoms. They, however, are usually very much like those of cerebral anæmia. They consist in a sense of fullness of the head, headache, dizziness, sometimes faintness and there may be dullness and drowsiness, irritability and depression, in fact, all such symptoms as are found in slowly acting, badly nourished or toxic brains.

Cerebral anæmia is a more definite pathological condition. Acute anæmia of the brain occurs as the result of severe general hemorrhage, or of sudden intra-cranial pressure as in cerebral hemorrhage, and of acute serous effusion. It may be caused by a weak and diseased heart. It occurs from sudden dilatation and paralysis of the vaso-motor splanchnic area, and it probably occurs sometimes in cases of extreme fright. It is caused also by certain toxic conditions, as in the vaso-motor paralysis of chloroform poisoning. It may occur in conditions of impeded circulation of the lungs and in mechanical obstruction in the flow of blood in the neck.

When the condition is of acute onset, there is dilatation of the pupils, respiration deepens and is irregular; there is dizziness, slowness of the heart beat and rise of arterial pressure. This is followed by a rapid heart beat, rise of arterial pressure and finally respiratory paralysis.

In very acute anæmia, such as is caused by ligature of all the arteries carrying blood to the head there is loss of consciousness and convulsions.

When the cerebral anæmia is of slow onset and more or less chronic in character, the symptoms are those referred to in large measure under the head of "passive hyperæmia." There is headache, tinnitus, vertigo, dullness, drowsiness, irritability, insomnia and depression. There may be dimness of vision and hearing, and muscular weakness and the general symptoms of a poorly acting brain.

As a matter of fact, however, it is probable that in cerebral anæmia these symptoms are due as much to the presence of toxic elements as to the insufficiency of blood.

In serious forms of general anæmia and even in the most severe types of pernicious anæmia no very marked cerebral symptoms occur.

CHAPTER XVIII

DISEASES OF THE BRAIN

These diseases, like those of other parts of the nervous system, consist of malformations, vascular disturbances, inflammations, softenings, hemorrhages, degenerations and scleroses, chronic infections, tumors and functional disorders.

THE INFLAMMATIONS OF THE BRAIN

The important forms are acute suppurative encephalitis, acute exudative forms of encephalitis, chronic meningo-encephalitis (general paresis) and multiple sclerosis.

ACUTE SUPPURATIVE ENCEPHALITIS

(*Abscess of the Brain*)

Brain abscess is a suppurative inflammation which affects the parenchymatous and other structures of the organ. It is always a focal disease, but may be single or multiple.

Etiology.—The primary cause of all forms of brain abscess is practically a microbic infection. The form of microbe, its mode of entrance, and the part of the brain attacked vary greatly. The predisposing causes relate chiefly to age and sex. Brain abscess rarely occurs before the first year or after the fiftieth year of life. It is rather frequent in young people, and occurs on the whole oftenest between the ages of ten and thirty. Males are more often affected than females in the ratio of about three to one. The exciting causes are chiefly disease of the ear, of the nose,[1] and of the cranial bones, injuries, remote suppurative processes, and septic and other infections. Chronic inflammation of the middle and internal ear is the most common cause of brain abscess, especially when that disease affects the tympanum and mastoid cells. Only a small percentage of abscesses (about 8 per cent.) follow acute otitis media. This is the reason more cases occur in adults. Caries of the ethmoid and nasal bones and of the orbital cavity leads to brain abscesses in a considerable proportion of cases. After chronic ear and bone diseases injuries are

[1] In 9,000 consecutive autopsies at Guy's Hospital there were fifty-seven brain abscesses due to ear diseases and one due to nasal disease (Pitt).

the most frequent cause, making up about 50 per cent. in all. The injury may be a compound fracture with direct infection from the open wound, or the abscess may be the result of *contrecoup* and may develop in a part of the brain opposite to that which was injured, or the abscess may develop below the point injured, there being apparently healthy tissus between the surface of the brain and the diseased part. These abscesses develop through laceration of brain tissue and subsequent infection of the wound with organisms. The most common remote suppurative processes which are followed by brain abscess are tuberculous inflammation of the lungs, fetid bronchitis, and empyema. Brain abscess may develop, however, from distant points of suppuration on the extremities or in almost any part of the body. General sepsis may lead to the production of brain abscess. Among the infectious fevers which are complicated with brain abscess are diphtheria, typhoid and typhus fevers, erysipelas, small-pox, the grippe. The oidium albicans or thrush may also be a cause. Brain tumors sometimes become surrounded by a suppurative encephalitis or may break down with the formation of mixed suppurative and neoplastic tissue. Tuberculous tumors are most frequently accompanied by suppurative encephalitis.

Symptoms.—Brain abscesses take sometimes an acute and sometimes a chronic course. In acute cases the symptoms develop rapidly and the disease runs its course in a few days or weeks. The symptoms come under the general head of those of pressure, those of poisoning from the diseased focus, and local symptoms due to irritation or destruction of certain special areas of the brain. The pressure symptoms are those of headache which is often very severe and persistent, vomiting which is quite frequent, though not invariable, vertigo, and a condition of mental dullness which may pass into a delirium ending finally in coma. Optic neuritis often occurs. The pupils are apt to be irregular, but furnish no definite indications. The pulse is usually slow, ranging from 60 to 70, but it varies a great deal. The temperature is normal or subnormal, as a rule, but this also varies, and it may rise several degrees above normal, always running an irregular course. The blood may show an increase in leukocytes and especially of the polymorphs. This is not always the case, however, and absence of leukocytosis does not exclude abscess. The toxic symptoms are those which we get in septic poisoning; namely, prostration, irregular fever, emaciation, anorexia, and such mental and sensory disturbances as have already been referred to. As a result of local irritation or destruction, there occur convulsions, paralysis, aphasia, and disorders of some of the special senses. Convulsions are not very common. When they occur they are generally of an epileptic character. The paralysis is usually in the form of hemiplegia. The cranial nerves are not often involved, if we except the optic. The urine is said to show

a diminution in chlorides and an increase in phosphates. The patient dies finally in coma from exhaustion.

In the chronic form of brain abscess the symptoms may for weeks, months, or years, remain practically latent, after the exciting cause has been at work and after the abscess has been established. The patient during this latent stage may suffer from headache, vertigo, mental irritability, and depression; he may at times have a convulsive attack. Occasionally there will be an exacerbation of the disease, at which time he suffers from intense pain, vomiting, perhaps delirium or a convulsion. From this he recovers and continues in a fairly good state of health again. After a variable period, usually of weeks or months, the terminal stage sets in. This terminal stage of the chronic form may assume very much the characters of the acute form already described. In other cases it shows itself by a sudden apoplectic or epileptic seizure or a sudden attack of coma, in which the patient sinks and rapidly dies. These terminal phenomena are due to the fact that the abscess, which has been previously encysted and quiescent, suddenly breaks into a lateral ventricle or through the surface of the brain, or to the fact that a hemorrhage occurs into the abscess.

Complications.—The common complications of brain abscess are a phlebitis of the superior petrosal and lateral sinuses and a meningitis. The phlebitis accompanies abscesses that are caused by disease of the ear. The meningitis may be caused by ear disease, but more frequently accompanies abscesses due to injury. When phlebitis is present there will be found an œdema about the ear and neck and a hardness of the jugular veins. In meningitis there is apt to be more rigidity of the neck, more pain, and there are often cranial-nerve paralyses.

Pathology.—Acute suppurative encephalitis resembles acute suppurative myelitis in the intimate nature of the changes that take place. There is an intense congestion of the part, which gives it a reddened appearance and which used to give to this process the name of *red softening*. This condition, however, is only the initial stage of the suppurative inflammation and does not deserve to be ranked as a special form of inflammatory process. As the process continues the parts become crowded with leukocytes and infiltrated with inflammatory exudate. The nerve-fibres and cells are destroyed, in part mechanically, in part by the poisonous influence of the pyogenic organisms. The nerve-cells lose their normal contours, swell up, and disintegrate; the neuroglia cells absorb the broken-down detritus and swell up, forming what are known as granular corpuscles; the leukocytes increase until a purulent mass is formed. The total result is a mixture of broken-down nerve-fibres and cells, leukocytes, and granular bodies. Bacteriological tests show the presence of various pyogenic microbes. Usually staphylococci or streptococci;

rarely the pneumococcus. The abscess thus formed varies in size from 1 cm. to 6 or 8 cm. in diameter (two-fifths of an inch to three inches). It is generally somewhat round, and if the case is chronic a fibrous wall is formed. It takes from three to four weeks for such a wall to develop (Fig. 201). Brain abscesses are usually single, occasionally there are two or three. In some conditions they are multiple, that is to say, there may be fifteen, twenty, or more. Multiple brain abscesses are always small and are usually due to pyæmic infection.

Location.—Brain abscesses involve the cerebrum oftener than the cerebellum, in the proportion of about four to one (Barr); two to one (Le

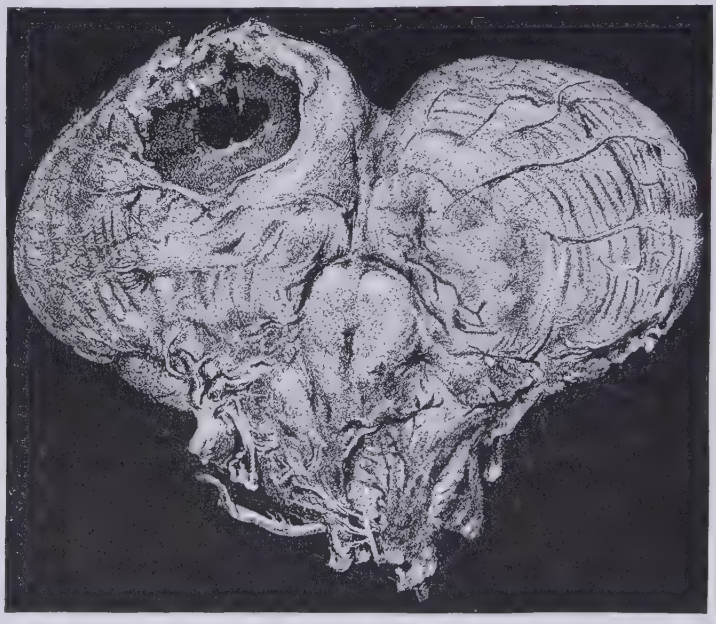


Fig. 201.—Abscess of cerebellum.

Fort and Lehmann). They occur rather oftener in the right cerebrum. They are very rare in the pons and medulla. The cerebral lobes oftenest affected are the temporal and frontal. In the cerebellum it is the lateral hemispheres that are most frequently attacked. The seat of the abscess has important relations to the cause. Abscesses due to ear disease are almost always either in the temporal lobe or in the cerebellum. If the ear disease is in the tympanum, the cerebrum is usually the seat of the abscess. If the disease is in the mastoid cells, the cerebellum is usually the part affected. If the disease is in the labyrinth, the abscess is also more apt to be in the cerebellum. This distribution of the seat of the disease is due to the anatomical relations of the bony parts to the temporal

lobe and cerebellum, respectively. Small veins from the temporal lobe and small veins from the tympanum connect with the superior petrosal sinus, and thus establish a pathway of infection. Often there is a sinus thrombosis and inflammation. Similarly small veins from the cerebellum and the mastoid and labyrinth connect with the lateral sinus, and here too a phlebitis may be established. Brain abscesses due to injuries are more frequent in the frontal and temporal lobes. What are known as idiopathic brain abscesses—that is, those which arise without any known cause—are most frequent in the frontal lobes. This is because most such cases are due to an unrecognized affection of the nasal cavities and ethmoid bone. Infection from the nasal and ethmoid cavities may lead to sinus thrombosis also, or to a localized purulent meningitis, some-

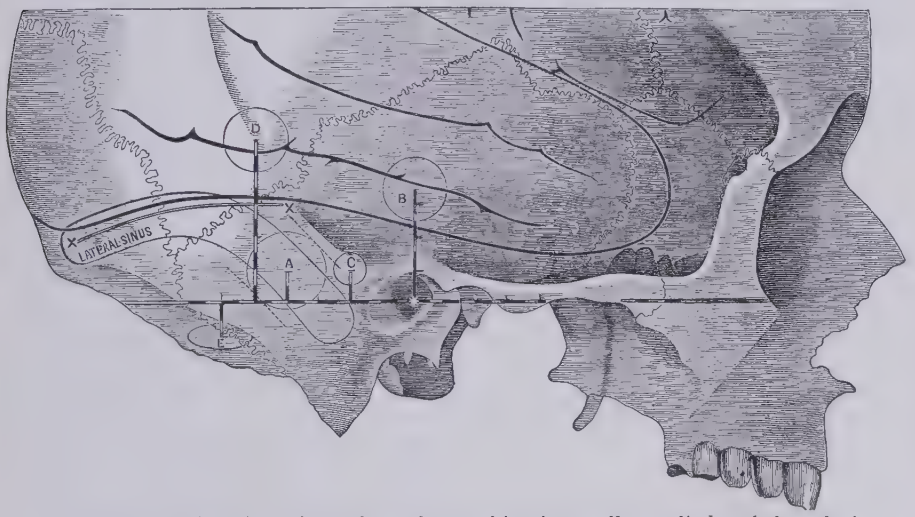


FIG. 202.—Showing the points where the trephine is usually applied and the relations of the sinus. The divisions of the lines indicate quarter-inches. (*Lancet.*)

times called extra-cerebral abscess. Such infections sometimes cause also only a local non-purulent meningitis involving the optic nerves. Brain abscesses due to suppurative processes in the lungs and pleura are probably embolic; and, as the emboli are carried up into the middle cerebral artery, the brain abscesses having this origin are situated in the field supplied by this artery. In children under ten, in whom brain abscess is usually due to ear disease, the cerebellum is more apt to be affected.

Course.—Acute abscesses last from five to fourteen days, rarely over thirty days. Traumatic cases run the shortest course. Chronic abscesses may have a latent period of weeks, months, and in rare cases even one or two years. When terminal symptoms come on, death occurs in a

few days. In a few cases brain abscesses have been spontaneously evacuated through the nose. Aside from this, the termination is always a fatal one unless surgical interference takes place. There is sometimes a recurrence of the abscess after an operation.

Prognosis.—The outcome of brain abscess is always a fatal one unless surgical interference takes place. There is sometimes a recurrence of the abscess after an operation.

Diagnosis.—The diagnosis of brain abscess is based upon the history of injury, aural or nasal disease, or remote suppuration, upon the general symptoms of sepsis, upon the presence of headache, vomiting, slow pulse, normal or subnormal and irregular temperature, a local tenderness of the scalp and rise of temperature over the seat of the lesion, hebetude, delirium, optic neuritis, rapid wasting, and diminution of chlorides in the urine and leukocytosis with a high polymorph. count. Lumbar puncture may help to exclude a meningitis. Puncture of the brain itself has been recommended, and it may be tried under conditions which allow prompt operation if indicated.

The diagnosis of the location of the abscess is based upon the history of its cause, whether from injury, ear disease, emboli from the lungs, or nasal disease; also upon the presence of hemiplegia, local convulsions, tenderness and rise of temperature of a certain area of the scalp. As brain abscesses are apt to affect latent regions like the temporal and frontal lobes, local diagnosis is often difficult. The diagnosis must be made from tumors of the brain, meningitis, and phlebitis of the sinuses. The differential points are given under the heads of the diseases mentioned. In some cases of otitis media symptoms of abscess occur, but there is an exquisite superficial tenderness limited to the region of the trigeminus. These cases recover and are perhaps due to a dural congestion (Cushing). Sometimes, also, in otitis symptoms of the acute serous meningitis of Quincke occur, which are relieved by lumbar puncture. Sometimes with nasal disease there may develop optic neuritis, headache and other symptoms of brain involvement, but no general symptoms, and no symptoms of serious brain irritation. These are cases of localized frontal meningitis and they get well.

Treatment.—The actual treatment of a brain abscess after it has developed is, as already stated, exclusively a surgical one. The successes so far have not been very great, but they have been sufficient to justify operation and to furnish greater hope for the future, when a more exact diagnosis can be made and a wider surgical experience has been obtained. According to Cushing, the percentage of recoveries now is about fifty. The accompanying figure shows the points to be located in trephining for abscess from ear disease. Something is due to the patient in the way of prevention, especially in cases of persons who have chronic aural or

nasal disease with carious processes. These should be carefully watched and treated.

ACUTE NON-SUPPURATIVE ENCEPHALITIS

(*Hemorrhagic Encephalitis, Hemorrhagic Polioencephalitis*)

Acute non-suppurative encephalitis is an exudative and sometimes hemorrhagic inflammation of the brain, characterized by symptoms of general infection or toxæmia, of severe cerebral irritation, and the signs of local cerebral lesion. It is sometimes spoken of as a non-suppurative encephalitis and as a curable encephalitis. The disease occurs as one form of the infection when an epidemic of anterior poliomyelitis occurs. In some of its forms, it is probably a rather common affection, complicating especially the acute infectious fevers of childhood and of influenza.

The disease then may be grouped in the following manner:

Acute non-suppurative encephalitis.

1. *Heine Medin type* of encephalitis due to the microbic infection which causes infantile paralysis. It is the encephalitic expression of an infantile paralysis infection.
2. *Acute encephalitis*, due to various other infections.
3. *Polioencephalitis superior and inferior*, due to influenzal and other infections.

1. When the inflammation is due to the poliomyelitic infection it occurs as a complication of the myelitis, as in Landry's paralysis; or it may occur as a separate focal lesion. Such cases have been noted in epidemics. Fortunately the complication is rare.

2. The ordinary type occurs rather more frequently in children, and a special children's type has been described. It attacks the cerebral hemispheres, sometimes the cortical gray matter alone, very rarely the cerebellum.

3. The mid-brain, pontine and bulbar forms are seen in the adults, most frequently in association with alcoholism and influenzal infections.

Etiology.—The disease is caused by the same infection as that of epidemic poliomyelitis; by other acute infections, especially those of influenza, of the fevers of childhood, of otitis media, and other nearby infected centres; it is one of the results and complications of serious alcoholism. It occurs most often in the young—except in Type 3 caused by alcohol and influenza. In some cases, trauma, when associated with some slight infection and perhaps laceration of the brain, causes it. Encephalitis has been known to follow a malignant endocarditis, and in connection with the infections of the puerperal state.

Symptoms.—Symptoms of the disease, when it affects the medulla or mid-brain (Type No. 3), are described under the heads of *acute bulbar palsy* and *acute ophthalmoplegia*. When the disease attacks the hemispheres, it begins, as in most of the cases, rather suddenly and without notable premonitory symptoms. The patient is seized by headache followed by fever sometimes reaching 105°F. This may be associated with vertigo, vomiting, photophobia and delirium. In children there may be convulsions. The symptoms of irritation disappear and are followed by a condition of semicoma or stupor. The patient can generally be partly aroused, and he does not usually have the stiff neck or the small pupils of meningitis. The respirations are shallow and frequent, the pulse is rapid and feeble. As the disease progresses, the deep reflexes are diminished and later the sphincters may be involved. After the patient has lain in a semicomatose condition for several days, he may become less stupid and more irritable and restless; or after two or three weeks of comparative stupor he may begin gradually to improve and, in a few weeks more, convalescence takes place, leaving him well or with some motor or mental defect.

In some cases an epileptic convulsion may occur in the early part of the disease. Again, as the disease develops, aphasia and paralysis of the arm or leg, or hemiplegia, may appear. In accordance with the location of the inflammation, the patient may have disturbances in the motor sphere, or he may have hemianopsia, hemiataxia, or disturbance of the cranial nerves, such as nystagmus, or eye palsy, or difficulty in speech and deglutition. Children may be left with a mental defect, and probably mild attacks of this disease are often the cause of such defects, or of epilepsies, attributed, perhaps, to an ordinary infective fever. An optic neuritis may also occur.

Course and Prognosis.—The disease is always serious but is not by any means always fatal. It may in its milder form run a course of two or three weeks, the patient gradually coming out of his stupor and making a slow recovery. In other cases the coma continues to deepen and the patient dies of exhaustion, and in still other cases the disease passes into a chronic state in which he lingers for weeks and even months.

Diagnosis.—The Heine-Medin type of encephalitis may be suspected if the symptoms develop in the course of an epidemic of poliomyelitis. The third or alcoholic and influenzal type of the disease occurs in adults. The second or common type is seen oftenest in children and is probably often mistaken for meningitis. It is to be differentiated from this by the sudden onset with coma, the absence of projectile vomiting, and of pinhole pupils, stiff neck, hyperæsthesia, and rigidity of the limbs. The presence of hemiplegia or local paralysis, or the occurrence of an epileptoid attack would point to encephalitis. The previous occurrence of an attack of

grippe in the young, or of exposure to the sun or acute alcoholism in the adult would lead to the probability of an encephalitis. By the help of lumbar puncture fluid can be withdrawn from the spinal sac, and examination of it will exclude meningitis.

Pathological Anatomy.—The pathological process underlying this disease consists of an acute inflammation with intense congestion and numerous small hemorrhages and capillary emboli. There is some hemorrhagic exudation as well as infiltration of leukocytes, with a certain amount of softening of the cerebral tissue in the neighborhood. The parts most frequently affected are variously stated to be the frontal and occipital lobes, the central convolutions, the semi-ovale, the temporal lobes, the base of the brain, and the corpus striatum. In four cases which I have examined, the process was in the temporal lobes, the parietal lobule, the mid-brain and the corpus striatum. It has been known to affect the cerebellum. If the process is a mild one, the hemorrhage and exudate are absorbed, and the injured brain tissue is gradually replaced by connective and neuroglia tissue. In this way small foci of sclerotic tissue are formed and the patient may afterward suffer from symptoms due to this condition. In the severer cases the softening becomes more extensive, larger hemorrhages occur, and in one case I have seen a massive apoplexy as the terminal stage.

Treatment.—The patient should be kept quietly in bed and should be given an active purge. Calomel is usually employed, but croton oil has seemed to me to be much more efficient as an eliminative and counter-irritant. The kidneys should be kept active, an ice cap placed upon the head, and leeches placed at the back of the neck. Urotropin may be given. The treatment after this can be only that of elimination, sedation and support. If the patient is stuporous and has a high fever small doses of aconite should be given. If he is asthenic, he should receive strychnine. The nourishment should be carefully attended to and, if he suffers pain, he should have morphine. Chloral and bromide seem to be the most efficient agents for relieving the restlessness and insomnia.

GENERAL PARESIS

(General Paralysis of the Insane—Dementia Paralytica)

General paresis is a disease of the brain characterized clinically by peculiar associated physical and mental disturbances, which end eventually and usually in general paralysis and mental deterioration; anatomically it is characterized by a syphilitic parenchymatous meningo-encephalitis. The disease cannot always be distinguished clinically from meningo-vascular syphilis.

Etiology.—Paresis, is a disease of modern civilization and of syphilization. It was a medical curiosity a hundred years ago; now it is extremely

frequent in our asylums, in neurological clinics, and in private practice, and it has become much more common of late years partly because it is more frequently recognized. It is found in all the civilized races of Europe and America. It affects even the inferior races living among civilized people, and is found, for example, among the negroes of the United States, affecting especially the negresses (Dieffenbach).

It is said to be rare among Chinese and Japanese and in Mohammedan countries even where syphilis prevails. It is more frequent in countries where social conditions lead to the spread of syphilis. It is rare, as a rule, in rural communities as compared with cities. However, in countries where there is enforced military training and a period of barrack life, the disease becomes prevalent in the country. It occurs oftener in men than women. In my private practice the ratio is about 10 to 1; in hospital practice it is about 3 or 4 to 1. It occurs oftener in the married absolutely but not relatively. It is a disease of middle life, most cases occurring between thirty and fifty; and few cases develop before or after these ages. The women when attacked are a little younger than the men.

Neuropathic races and neuropathic persons are more susceptible to the disease. Paresis is sometimes (0.03 per cent.) directly transmitted. It then appears between the ages of ten and twenty and is known as juvenile paresis. In this form males and females are about equally affected.

Occupation is a predisposing cause in just about the proportion with which the occupation leads to exposure to syphilis. In Europe it is very frequent among military officers in some countries and it is very rare among the Catholic clergy.

The excessive use of alcohol, excessive mental exertion, if combined with emotional strain and excitement, sexual excesses and abuse are predisposing causes. A certain popular idea that the disease is the result of perverted sexual indulgence is not correct. It has now become firmly established that paresis is always due to a syphilitic infection, which leads to a parenchymatous degeneration of cerebral neurons; just as tabes is due to a similar affection of the spinal neurons. It occurs in about 1 to 2 per cent. of all luetics and makes up about 10 per cent. of our asylum population. It comes on oftenest between the tenth and twentieth year after the infection.

Symptoms (*Prodromata*).—Paresis is sometimes preceded by a prodromal period which may last for four or five years, more often perhaps a year. During this time the patients suffer from symptoms that resemble somewhat those of neurasthenia, sometimes those of a mild anxiety psychosis. The patient shows a change in character. He is more irritable, less able to concentrate, has some memory defect, and is less efficient

in his work. He has headache, insomnia and cerebral and spinal dysæsthesias, and paræsthesias. He worries about himself and is termed hypochondriacal. At this stage he often has quite clear insight and appreciates his danger. But unlike cases of true neurasthenia he cannot do efficient work, though he may wish to do so. If there is a little temperamental elation he may work unwisely and foolishly. It sometimes happens that the peculiarities of mind and conduct are so slight that, it is only in retrospect, and after serious conditions develop that the family remember the early changes.

In more advanced cases the patient may suddenly develop a convulsion, or some change in the pupils and reflexes may appear.

Physical examination may not reveal any objective changes, except that his fluids may show positive reaction to the Wassermann and other luetic tests, but this is rather easily controlled by treatment. Such patients under active treatment may get well; but if the trouble is not recognized, some form of cerebral syphilis eventually develops. Even if this luetic neurasthenia progresses till some characteristic mental and physical symptoms of paresis or tabo-paresis, or meningeal syphilis develop, the patient may get well or at least secure a complete remission. The preparetic condition is a curable one.

But besides these neurasthenic and anxiety prodromata, paresis may be preceded by a distinct functional psychosis. This takes the form oftenest of a luetic melancholia, more rarely of a mania. The patient may and generally does emerge from this and he may then remain well, but more often unless treatment is unusually effective, he begins to develop the characteristic signs of general paresis. Very rarely paresis comes on suddenly like an explosion.

Finally and not rarely paresis is preceded by distinct symptoms of cerebral or cerebrospinal meningeal syphilis, or by a tabes dorsalis.

Thus paresis is approached in four different ways, clinically speaking:

1. By a luetic neurasthenia, or anxiety psychosis.
2. By a luetic melancholia or mania.
3. By a meningeal cerebral or a spinal syphilis.
4. In a gradual or explosive manner.

General paresis is classified in various and numerous ways. Many descriptions have been based on a study of custodial and more or less advanced cases.

The description and classification I give are based largely on a personal study of cases as I have seen them develop and pass into hospital conditions or into states of improvement and remission. The various phases of advanced paresis have little practical interest.

The disease develops in

1. An excited form.

2. A depressed form.
3. With quiet dementia.
4. In an explosive form.

These various types may change in the course of the disease.

In my experience about one-third are at first seriously depressed, less than a third maniacal and more than a third pass with perhaps some slight excitement or delusional activity into a progressive dementia; that is to say, unless the course is modified by treatment. In all cases some meningeal symptoms may be present.

Symptoms of Onset: Active Type.—In the first type of cases the patient begins by showing unusual irritability of temper; trivial things annoy him, and his bad humor and change of disposition become noticeable in his family relations and in his business. He is fretful; complains of being easily fatigued; loses interest in his affairs, and is unable to fix his attention for any length of time upon them. He makes occasional mistakes of judgment, and does some extravagant or foolish thing in the way of purchasing or selling. He is totally lacking in insight. This condition of irritability is followed by one of great mental exaltation. The patient becomes very happy and cheerful and confident; he feels better than he ever did before in his life. He talks excessively, and is effusive and jocose when he used to be sober and reserved. He develops great schemes for the future, he lavishes money uselessly in making presents to his family and friends, or in some extraordinary business venture, and imagines himself possessed of immense wealth or great power. He has, in other words, delusions of grandeur, or megalomania. This condition of exaltation is interrupted by outbursts of violence, especially if it leads him to indulge in drink, as is often the case. In the course of three or four months the symptoms become so marked that the family recognize the seriousness of his state, and he is confined in some institution where he can do himself and others no harm. Under institutional *régime* he now becomes somewhat more quiet; his exaltation softens down. His symptoms may even remit, and for a time he becomes nearly or quite rational. But after some months he begins to show distinct signs of dementia; the memory becomes weak, he forgets recent events, mislays things, makes mistakes in his accounts, is unable to add correctly; he cannot write a letter coherently, or if he does there are mistakes in spelling and elisions of letters.

During or before the exaltation there gradually appear physical symptoms which are very characteristic. The patient's hands become tremulous, and his handwriting is so affected that his signature often cannot be recognized. There is distinct and decided facial tremor, particularly apparent if the patient is made to close the eyes and stretch the muscles of the lips so as to show the teeth.

There is marked tremor of the tongue, all this tremulousness being much more exaggerated than is seen in other diseases, except occasionally in acute alcoholism. The speech becomes stuttering and thick, and he cannot pronounce long words clearly. On examination of the reflexes it is usually found that the knee-jerks are exaggerated. The pupils are almost always uneven and, as a rule, react badly to light though fairly well to accommodation, showing, in other words, the Argyll-Robertson pupil; sometimes they do not react either to light or accommodation. The fundus oculi is normal.

There is an early and decided weakness of the sexual function. The bladder may also become weak. The appetite and vegetative organs remain in fairly good condition. The patient often suffers from persistent insomnia. During this time he also has occasionally vertiginous, syncopal, or apoplectiform attacks. In the latter he falls down and perhaps has hemiplegia lasting for a few days or a few weeks. An epileptic convulsion may occur.

In some cases the knee-jerks are abolished, and there is some ataxia and evidence of a posterior sclerosis. The general muscular power is much diminished, and the patient is unable to take long walks or do any great amount of physical exercise.

In the second stage the most striking feature is the gradual onset of dementia. The patient now becomes more quiet and is inclined to sleep during the daytime. He takes little interest in affairs about him, is extremely forgetful, and is often unable to recognize even his intimate friends. He no longer knows the day of the month nor the year, and cannot tell one anything about current events of the day. He becomes gradually careless about his person, and has to be watched while at his meals lest he spill food on his clothes, and at the toilet lest he soil himself. Finally, he needs to be cared for as if he were a child.

At this late period, again, attacks of an apoplectiform character may come on, leaving him temporarily or perhaps permanently hemiplegic. His appetite often continues good, sometimes voracious, and he may gain flesh. He is apt at this time to have periods of excitement in which he has delusions of persecution, or he may have some slight delusions of grandeur. One patient of mine used to weigh himself every day and think he was gaining ten pounds each time. He kept on till he thought he weighed nine hundred pounds.

In the last scene of all he becomes bedridden and helpless, terminal febrile attacks occur, and he finally dies of exhaustion. The somatic symptoms during this last period consist of increased tremor, disturbances of speech, and gradual muscular weakness until the patient becomes helpless.

The average duration of the disease is about three years. There

are some acute, galloping forms in which the patient dies within a year, and there are some cases in which the patient reaches a stage of partial or complete dementia and remains in this condition for ten or fifteen years.

The Depressed Type.—In this form the disease begins with symptoms resembling those of neurasthenia and hypochondriasis. The patient complains of disagreeable sensations about his head, hemicrania, pain in his limbs and back, inability to sleep, disorders of the stomach, and vague sensations of discomfort and oppression which he is unable distinctly to describe. These are the patients who are often treated as neurasthenics for a long time, and at first show hardly any physical or mental symptoms suggestive of the real trouble. Careful examination, now, however, will show some objective physical changes including a positive serological formula.

Now evidences of mental disturbances appear, and they are mostly those of dementia with perhaps delusions of persecutions and suspicion. These delusions may be accompanied by occasional outbreaks of excitement and violence, but the paretic is rarely homicidal and, it may be added, rarely suicidal, except when he has distinctly a melancholia. After dementia has set in, the physical symptoms of tremor, scanning speech, and tremulous handwriting all become noticeable, and the final stage resembles that of the other form.

Dementing Type.—In a third type there is a primary dementia. The disease begins without any excitement or any special depression, with symptoms of forgetfulness, lack of attention to business, and incapacity to do work. The patient makes mistakes in his calculations, mislays and forgets things, and soon is found by his employer to be of no use. He is often good-natured, not unhappy, and without distinct delusions of any kind. The somatic symptoms of tremor, fixed pupils, and exaggerated reflexes may appear, but sometimes are entirely absent. Scanning speech is not always present, or comes on late.

Tabo-paresis.—Some symptoms of tabes are present in almost 10 per cent. of paresis. It is the paresis that is the early, dominant and important factor; for tabo-paresis is really and practically paresis. The tabetic symptoms are of the minor kind: Argyll-Robertson pupil, lightning pains, loss of knee-jerk, some hypotonia and more rarely some ataxia. When tabes becomes fully and first developed, paresis rarely appears (3 to 5 per cent. of cases).

Meningo-vascular or Pseudo-paresis.—There are some cases of paresis in which symptoms of exudative syphilis introduce and accompany the disease. The patient has at first eye palsies or attacks of hemiplegia with intense headache followed by convulsions. It is recognized that he has a syphilitic exudate pressing upon some part of the brain, either

the base or the convexity, usually the former. Under proper treatment, this resolves and he gets over the paralysis and the seizures, but it is now found that his mind is slightly affected. He has no delusions perhaps, and no immediate exaltation, but his memory is impaired, judgment weakened, his emotional condition is one of excitability, and he has to give up business and live a quiet, inactive life. If he does this, in some cases the disease becomes arrested, and he remains fairly well for a number of years. Cases of apparent recovery have been reported, but unless treatment controls the situation, dementia finally sets in. In these cases there is both a parenchymatous and a meningeal syphilis.

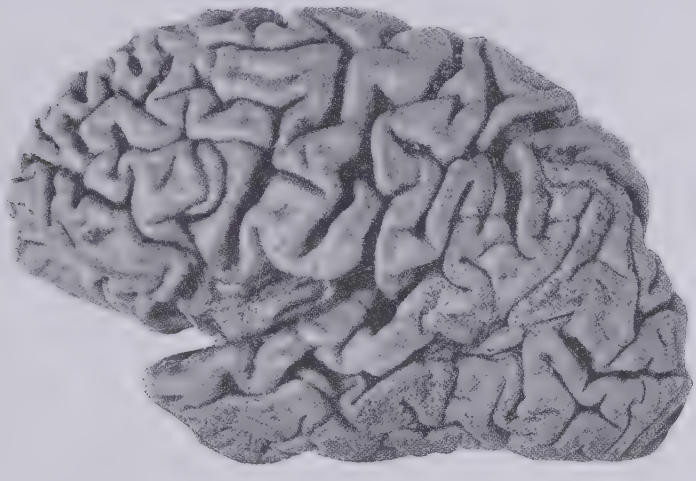


Fig. 203.—Left hemisphere in a case of general paresis. The præfontal area of higher association is grossly wasted. The psychomotor area shows some but much less waiting. The temporal, parietal and insular regions of associations are acutely changed. The after part of the temporal region of association and the anterior part of the parietal show less acute change and more wasting. The visual center and the visuopsychic cortex around it are intact. (*Bolton.*)

Alcoholic Pseudo-paresis.—Persons who have for long periods of time continuously and excessively indulged in alcohol may develop, and generally do, a condition of mental weakness which to a certain extent simulates paresis. If these patients have not had syphilis, however, it is not a true paresis. The patients become weak-minded, feeble in judgment, poor in memory, their moral instincts get out of control, and acts of extraordinary selfishness, bestiality, and besottedness are manifest. They gradually become more enfeebled, and finally enter a condition of dementia if they are not carried off by some intercurrent disease, as is often the case. These cases, however, do not present the physical symptoms of paresis. They do not have the speech disturbances, the paralyses, or the apoplectiform seizures that

characterize the true disease, and if they can be kept from alcohol they may remain in a state of partial dementia for many years.

Diagnosis.—The diagnosis is based on the history of syphilis, the positive serology, the facial and tongue tremor, irregular and rigid pupils, exaggerated or irregular knee-jerks, the disturbance of speech, weakness of memory, loss of power to work, irritability and change of character, change in the handwriting, and presence of convulsive or apoplectiform attacks. Examination of the cerebrospinal fluid and blood shows serum

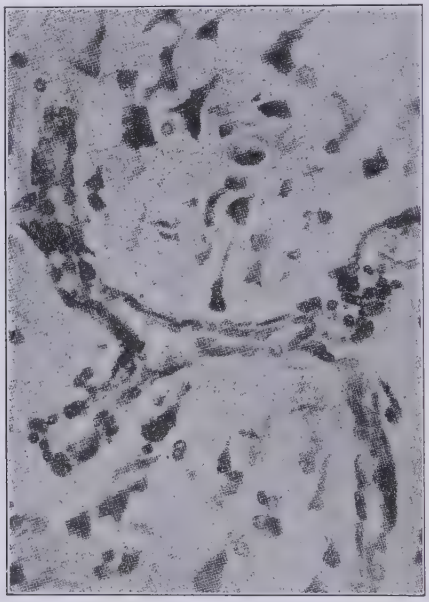


FIG. 204.—Early general paresis. A small cortical artery is shown with a beginning injection of round and plasma cells; dying pyramidal cells are also to be seen (×320). (*Bolton.*)

Wassermann+, cerebrospinal fluid, Wassermann+, cells per cubic millimetre++, globulin+, colloidal gold+ (see table, page 412).

Pathological Anatomy.—The dura mater is adherent to the calvarium in places or throughout. In old cases there is sometimes a pachymeningitis interna with hematoma. The pia mater is cloudy and thickened, especially over the frontal and parietal regions, less on the base and never over the occipital lobes. The brain is atrophic in old cases, with shrunken convolutions and local areas where the brain has especially atrophied. The ventricles are dilated and show a granular surface. The loss of brain weight averages about five ounces and is most in the frontal lobes. Microscopically the cortex is irregularly thinned, and the arrangement of cell layers is distorted. The third and next the

second layers are the most affected. The nerve-cells show all stages of degeneration. The cortical fibres also are atrophied and in the last stages of the disease have quite disappeared, the supra-cortical radial fibres being the first to go. The neuroglia tissue is greatly increased and shows evidence of active proliferation and finally forms sheaths about the blood-vessels. (Fig. 205.)

The blood-vessels are especially affected. There is an active formation of new vessels, with numerous dilated capillaries and widened adventitial lymph spaces. These spaces are filled with cells which are partly lymphocytes and partly plasma cells. This vascular change,

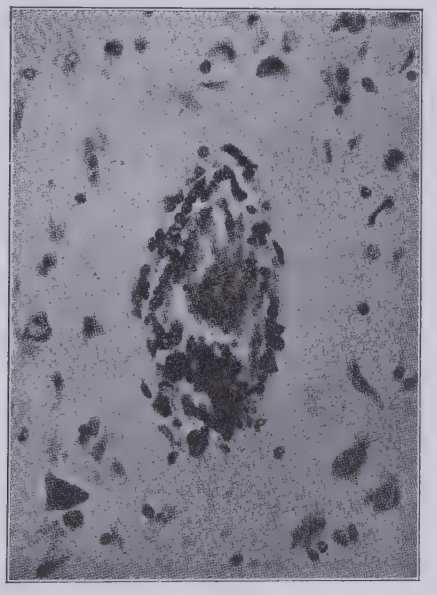


Fig. 205.—Very advanced general paresis. A vessel of the gray matter showing partially organized periarteritis, and outside of this a neuroglial sheath (× 320). (*Bolton.*)

with plasma cells, is one especially characteristic of paresis (Vogt, Alzheimer). The cells are large, round or irregular in shape, with a protoplasmic body. It is not known whether they come from connective tissue or the leucocytes of the blood.

In other parts of the brain focal, diffuse and tract degenerations occur. The cerebellum is affected like the cerebrum but to a less degree. The spinal cord shows degenerations in both the posterior and lateral columns in about two-thirds of the cases. Some degenerative changes occur in the sympathetic system and peripheral nerves. The *treponema pallida* has been found deep in the cerebral tissues, but the organisms are few in number and difficult to detect. The explanation of how they

cause such an extraordinary disintegration of the master tissue of the body makes a long chapter in pathology, and one not yet completed.

Prognosis.—The prognosis was formerly thought to be invariably bad. If one sees the patient, however, in the earliest stage, removes him at once from sources of excitement, and makes him live quietly, using anti-luetic and tonic treatment, one can sometimes check the disease, and practically cure it. When the disease has well entered upon its course it is incurable by any measures as yet known.

The prognosis is better the more the symptoms and serological formula suggest meningeal syphilis. It is better in cases in which the patient shows "insight" as to his condition; better when the mental symptoms indicate a functional psychosis more than a deterioration. Speech defects are of unfavorable omen. Tabo-paresis has about the same prognosis as paresis. When meningo-vascular syphilis exists, if a tabes develops paresis does not, as a rule, and the outlook is not so serious. Too much importance may be placed on the serological formula. It may be partly positive and yet the patient may do very well. Positive reactions may disappear under the influence of time and good hygienic conditions alone.

Laboratory Tests for the Presence and Differentiation of Syphilogenous Nervous Disease.—The serological formulæ obtained by testing the blood serum and the cerebrospinal fluid is here given for the three important luetic nervous diseases—meningo-vascular syphilis, tabes dorsalis, paresis and tabo-paresis. This table was prepared by Dr. David Kaplan of the N. Y. Neurological Institute, and is the result of over two thousand complete examinations. Many of the fluids tested were from my own patients. I have had occasion to follow this work and the formulæ and conclusions given agree with my observations up to the present time.

THE SEROLOGICAL FORMULÆ FOUND IN SYPHILITIC NERVOUS DISEASES

	Cells per cc.	W.R. in C.S.F.	Globulin.	Fehl. Reduct.	Colloid. Gold	W.R. in Serum
I. *Meningo-vascular Lues* (Cerebral, Spinal or Cerebrospinal).	Average 80 to 200 Extremes 0 to 2000	Plus in 65 per cent.	Excess in 65 per cent.	Present in 95 per cent.	Present less than 2 per cent.	Plus in 80 per cent.
II. *Tabes Dorsalis*.....	Average 35 to 60 Extremes 0 to 350	Plus in 40 per cent.	Excess in 33 per cent.	Present in 99 per cent.	Absent	Plus in 65 per cent.

THE SEROLOGICAL FORMULÆ FOUND IN SYPHILITIC NERVOUS DISEASES.—*Continued.*

	Cells per cc.	W.R. in C.S.F.	Globulin.	Fehl. Reduct.	Colloid. Gold	W.R. in Serum
III. *Paresis*	Average 18 to 35	Plus in 85 per cent.	Excess in 75 per cent.	Present in 100 per cent.	Present in 95 per cent.	Plus in 95 per cent.
and	Extremes 0 to 250*					
Tabo-paresis	20 to 40	Plus in 85 per cent.	Excess in 70 per cent.	Present in 100 per cent.	Present in 75 per cent.	Plus in 95 per cent.

The Wassermann reaction on the spinal fluid was performed by using 0.5 cc. of the medium, with two antigens; one containing cholesterin.

The globulin content was determined by the butyric acid ammonium sulphate method. The cells were counted by the Fuchs-Rosenthal method, the same day the fluid was obtained.

Meningo-vascular Lues.—This form represents the serology of the earliest involvement of the nervous system. The increase in cells may appear before a positive Wassermann reaction or a globulin excess, and may persist until cerebrospinal functions are impaired and detected by the neurologist. The cell count, which is the chief feature of this pre-eminently exudative process, may reach into the thousands, but is very readily influenced by treatment. The positive fluid Wassermann reaction disappears in such a case very quickly, and if a great many cells of the polynuclear variety are present, thus giving rise to the absent Fehling reduction, the latter returns also with the fall in the pleocytosis (increase in cells). The majority of cases with this form of luetic nervous involvement show on an average from 80 to 180 cells per cubic millimetre after moderate therapy. In this form of lues of the nervous system one is able to do away entirely with abnormal findings in less time and with fewer remedies than is the case with tabes or general paresis. Some cases remain uninfluenced after the cell count reaches 20 to 30 per cubic millimetre, using the intravenous method of salvarsanization. In case the intra-spinous route is selected, the pleocytosis is influenced more radically on a mechanical basis, depending chiefly upon the removal of the cells as one would evacuate an abscess. This is further corroborated by the persistence of the globulin excess and the positive Wassermann reaction which are to be regarded, therefore, as an expression of organic response not depending *in toto* upon the local disease.

Tabes.—The early forms of this disease will give a serology closely resembling that of the meningo-vascular form of lues. The cell count will then show its upper limit, the globulin will give an intenser precipitation than in the later stage, and the Wassermann reaction will be positive in both media. As the disease progresses and assumes its more or less permanent features, the serology also tends to settle to what constitutes the type of tabetic serology. The Wassermann reaction in the serum is positive, that of the fluid is negative, or only weakly positive, the excess of globulin is less marked or entirely absent. As the disease

progresses and enters the final state, the entire serology may become negative. Unless a paretic accompaniment is detected, the above holds true for the majority of tabetics. If, however, paresis becomes an accompaniment, the serology tends to assume also the serological features of that disease.

Therapy will find an obstacle in some cases in being unable to render negative a positive serum Wassermann (the "Wassermann fast" phenomenon). The Wassermann reaction in the fluid will also manifest itself together with a comparatively low cell count, and in a small number of cases one will be able to obtain the characteristic curve with the colloidal gold as found in general paresis. The latter form is entirely different from the form closest to the meningo-vascular type, in that the serology is easily rendered negative, and the high cell count (hyperlymphocytic tabes) will be found to fall with the very first salvarsan injection, although given intravenously. The fall may be a few hundred cells to the cubic millimetre.

General Paresis.—Aside from furnishing the greatest number of cases that show pathological fluid and serum changes, this disease also furnishes the various abnormal constituents in its greatest intensity. The "Wassermann fast type" is a feature of this disease more often than in the other forms of lues of the nervous apparatus. It also shows to the exclusion of the other forms of luetic disorder, the characteristic precipitation of colloidal gold, which is one of the most constant accompaniments of this disease. In fact where one is able in rare instances even to influence the Wassermann reaction, one will find that the colloidal gold phenomenon still persists. In early paresis one will occasionally find an absence of a positive Wassermann reaction. In the cerebrospinal fluid, the pleocytosis may also be very high, comparatively, showing thereby the common origin of all luetic nervous diseases, which start as a rule with a high cell count and probably with a meningo-vascular disturbance. The advanced form of paresis shows the full quota of abnormal findings, and as the degenerative process advances, one will sometimes miss the positive Wassermann reaction in the serum, while it may still be present in the cerebrospinal fluid. In the latter instance the cell count may be very near the normal cell count. In some cases only a few cells were encountered. That exceptions may be found in this form of neural lues must be admitted, so that one will here and there come across authentic paresis with a very high cell count, amounting to 200 or 300 cells per cubic millimetre; such extreme counts, however, are very rare.

KAPLAN.

Treatment.—The first and most important thing in the treatment of paresis in its early stages is to adopt measures for the treatment of syphilis

which is the underlying cause. Before the advent of the use of salvarsan this was done by the intensive use of mercury and iodides combined with eliminative measures, tonics and fresh air. These measures were not without distinct success and it even remains still to be proved that salvarsan treatment is more permanently efficacious than the older methods.

There has been of late years so much more interest and effort in the treatment of paresis that enormously better results are being obtained, apparently, by the new method, but certainly to some extent by reason of greater interest now taken in therapeutics.

The technical methods of using salvarsan and its preparations differ with different practitioners, and the final and best methods have not yet been agreed upon. The technic which the writer employs and which has been used to a large extent in the New York Neurological Hospital, is as follows:

The patient is given an intravenous injection of 0.45 neo-salvarsan. After an interval of two days he is given another injection of 0.9 and this injection is repeated every third day until he has had ten injections.

During the time that these injections are given he receives either a hypodermic of one grain of salicylate of mercury twice a week, or daily inunctions of mercury on the days on which he does not receive the intravenous injection.

After the patient has received the tenth dose, he is given a prescription containing gr. $\frac{1}{24}$ of bichloride of mercury and gr. x. of iodide of potassium and this he takes intermittently for the following four weeks.

After this he is given an intra-venous injection of neo-salvarsan every month for five months and then is given another intensive treatment, if the remission or improvement is not completely satisfactory.

The intra-venous injections are continued for a period of a year but with less frequency. They should be continued intensively irrespective of the condition of the blood and cerebrospinal fluids. The endeavor to measure the amount of treatment which should be given by the laboratory findings of these fluids is a mistake, or at least these things should not be too closely followed as therapeutic guides.

Naturally, while carrying out this technic, a proper regard should be had for the general condition of the patient's health, and measures such as laxatives, tonics, fresh air and rest should all be instituted.

The method thus outlined as a standard one needs variations in accordance with the state of the disease and its rate of progress. When there is evidence of an associated meningeal exudate the number of injections may be increased and even brought more closely together. I have seen a very good result from giving 0.9 neo-salvarsan daily for nine days. It seems that the nearer one can come to safe toxic doses the

better results will be achieved and for this reason it may be wise to give an intra-spinous injection of salvarsanized serum, or salvarsanized serum with the addition of neo-salvarsan itself in connection with this and in addition to the regular intra-venous injections. It may be necessary to give as many as thirty injections in the course of two or three months.

There is still a difference of opinion as to the relative value of salvarsan and neo-salvarsan, it being generally believed that the salvarsan is about twice as powerful as the neo-salvarsan, but that it does not act more beneficially, except for the fact that it is stronger.

The earlier in the course of the disease that treatment is given the better the outlook. On the other hand, in the third and terminal stage of the disease the treatment is useless and may even hasten the death of the patient. It seems wise in some more chronic cases to give a series of injections of mercury before giving salvarsan.

Opinions also still differ as to the value of the intra-spinous injections of salvarsanized serum (Swift-Ellis method) and of the intra-spinous injections of the patient's serum to which salvarsan is added. I have not seen any convincing evidence that the method is a superior one, but there are some who very strongly support it.

In the use of this method injections are given not oftener than once in ten days or two weeks, and the number of injections is generally determined by the effect of them upon the cerebrospinal fluid reactions. It has been found comparatively easy to reduce the number of cells to the normal amount in the cerebrospinal fluid by the various methods of treatment, but to make the blood and cerebrospinal fluid negative to the Wassermann reaction in true paresis is very difficult.

The result of treatment in early cases is almost invariably to secure a satisfactory remission, and these remissions in many cases are practically like cures.

The doctrine put forth by certain neurologists that when parenchymatous degeneration of the brain sets in, treatment is useless, is certainly untrue and not founded on any sound pathological basis. We know perfectly well that parenchymatous degeneration of locomotor ataxia is often arrested and patients live for thirty years or more. We also know that remissions have occurred in paresis lasting many years.

Aside from the treatment thus outlined the therapeutics of paresis is mainly tonic and symptomatic, in the second and third stages.

Various other specific treatments of paresis have been suggested and tried, such as the injections of nucleinate of soda and of tuberculin to produce artificial fever. I have not seen any successes from these forms of treatment.

Naturally the patient while undergoing specific treatment should be under close observation and be made to live a quiet and regular life. The influence of a life entirely in the open air is of great value in ameliorating the luetic infection. I have seen the serological formula become negative just by this means.

MULTIPLE SCLEROSIS

Multiple sclerosis is a chronic and intermittently progressive malady characterized by some paralysis, usually in the form of paraplegia, by coarse tremor, disturbances of speech, nystagmus, apoplectiform attacks, and various other cerebral and spinal symptoms depending upon the seat of the lesion. It is due to the development of sclerotic patches in the different parts of the brain and cord, which patches are for the most part the result of a neuroglia proliferation. The disease is probably an inflammatory rather than a degenerative one. It affects the spinal cord as well as the brain.

Etiology.—It occurs rather more frequently in the male sex and is a disease of the first half of life. Multiple sclerosis is, in fact, one of the few chronic nervous disorders of organic origin developing at this time. Most cases begin between the age of twenty and thirty. Cases have, however, been observed in infants and children, but the trouble in its typical forms does not appear in the declining years of life. While a few cases of hereditary multiple sclerosis have been reported, it is generally conceded that hereditary influence is very slight and even a hereditary tendency to degenerative disease or a neuropathic family history is rare though not unknown. Strumpell's theory that the disease is endogenous has not been sustained. There can be no doubt that the most important of all of the few known causes of multiple sclerosis is infection. This is so true that it may be called a post-infectious disease. The infectious disorders which are followed by sclerosis are typhoid fever, pneumonia, malaria, and the eruptive fevers. It has been known to follow also diphtheria, whooping-cough, erysipelas, dysentery, cholera, and even rheumatism. Another important cause is excessive muscular exertion, such as occurs in professional dancers, and amateur or professional athletes. Workers in metallic poisons, such as lead, copper, zinc, and even manganese may develop sclerosis. The influence of trauma in causing multiple sclerosis has to be very carefully weighed. It is certainly very rare, but there seem to be some few cases in which trauma appeared to be an exciting cause. It is generally accepted that neither syphilis nor alcoholism are causative factors, although syphilis may be associated with the disease and perhaps produce a peculiar luetic form of it.

The following data as to the frequency and cause of the disease were collected by Dr. C. W. O. Bunker, in his neurological prize essay:

Frequency.—Among 70,000 neurological cases in America the percentage of multiple sclerosis was 1 in 266, or 0.058 per cent. In Scotland it is about 2 per cent.; in Berlin, 0.85 per cent.; in Hamburg, 1 per cent.

Age.—The following table, compiled by Bunker and myself, represents American cases. It makes the incidence of the disease too late. Practically, the cases occur between the fifteenth and thirty-fifth years.

–10,	9
10–20,	17
20–30,	36
30–40,	45
40–50,	32
50–60,	24
60–70,	7
70–80,	4

The disease is twice as frequent in men as women—129 to 67; statistics of Jelliffe, Bunker and myself. European statistics make the numbers about even. The disease is seen more frequently in foreigners, perhaps, because our clinics are so largely foreign in population. My private cases are nearly all American.

Symptoms.—The disease begins insidiously. There may be a preliminary and rather short hemiplegic or paraplegic attack with some sensory disturbance. More often a comparatively short time after recovery from some acute disease the patient begins to suffer from weakness of the lower limbs with stiffness and some degree of heaviness, and numbness. The bladder is also a little weak, and it is difficult to retain the urine. These symptoms after a few weeks may disappear and the patient be well for months. If not, very soon the patient notices some unsteadiness in the gait, due not alone to weakness in the legs, but to an increasing degree of ataxia. He finds also that his hands are trembling and that this tremor increases upon voluntary motion. It is the type of tremor known as "intentional." He has at this time also some indistinctness in speech, it being difficult for him to enunciate long words. These come out in a slow, *syllabic* utterance, as it is called, each syllable being spoken separately. He may have also a little trouble in swallowing. By this time he has had some sensation of numbness in the limbs, and some pains occasionally in the joints and extremities, but the sensory troubles are not permanent or not very marked and are mainly paræsthesic. If he is examined now it will be found that the gait is stiff and awkward, the patient walking unsteadily; or, in other cases, it may simply be the stiff, weak gait of moderate paraplegia. The Romberg symptom will be found to be present to some extent. The knee-jerks are exaggerated and ankle clonus and Babinski's reflex may be present. The abdominal reflexes are absent early in many cases. The hands are

unsteady, and the movements are characterized by a jerky tremor, which may be so great that the patient has difficulty in dressing and feeding himself. This tremor disappears almost entirely if the patient lies flat upon his back. If he sits up, however, it may be seen perhaps in the muscles of the neck, causing the head to be oscillated, and constantly more or less regular tremor in the arms is present. As the disease progresses the speech becomes more thick, and often is almost unintelligible in the severer cases. Examination of the eyes shows a nystagmus, perhaps only when the eyes are turned to one side, but often the jerky movements are seen, even when the patient is told to look directly at

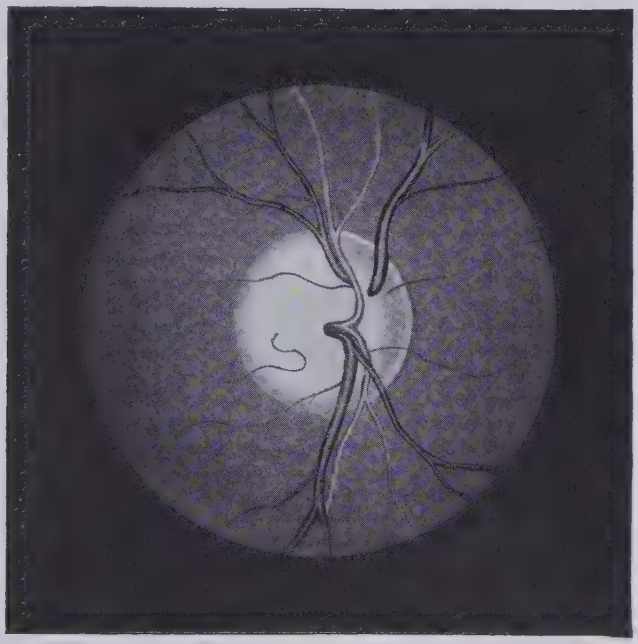


Fig. 206.—Pallor of entire optic disc except its nasal margin. (*Holden.*)

an object. Evidence of impaired eyesight appears with pallor of the temporal side of the optic disc (Fig. 206), and a central scotoma may be noted. The tongue is protruded in a jerky way, and attempts at swallowing are often awkward. If a glass of water is handed him, the patient seizes it, but in carrying it to his mouth he agitates it so violently that the fluid is spilled and perhaps the tumbler drops from his hands. Examination of the muscular system shows weakness of the legs, less of the arms, but no complete paralysis. There is no marked atrophy of the limbs, and no change of any moment in the electrical reactions. There are many cases in which the jerky tremor, syllabic

speech and nystagmus are not marked or develop very slowly, and more recent studies show that the most common symptoms are:

1. Bladder weakness.
2. Absence of abdominal reflexes.
3. Spastic paraplegia.
4. Optic atrophy.
5. Sensory defect (paræsthesiæ).
6. Sphincter trouble.
7. Speech defect.

Examintion of the cutaneous sense may show some little tactile anæsthesia in the limbs, but this is not always present and in general the cutaneous sensory disturbances are slight and evanescent. There is a certain amount of ataxia, which is not due to muscular anæsthesia but to inability to control and co-ordinate the movements. There is no loss of sense of weight or of pressure. Of the nerves of special sense, the eye is most frequently involved. The patient may have some diplopia from paralysis of one of the eye-muscles. The pupils react to light and accommodation. The involvement of the optic nerve may go on to an almost complete atrophy. On account of this the patient suffers from contraction of the visual field, central and other scotomata, and weakness of vision, but absolute blindness never occurs. Examination of the cerebrospinal fluid by lumbar puncture shows the absence of a lymphocytosis.

While the disease is running the course just described, the patient sometimes suffers from attacks of vertigo, and occasionally from sudden seizures resembling apoplexy, and even from epileptiform attacks. The mind is usually not much affected, and I do not recognize any special mental characteristics except a certain childishness of temperament. There may, however, be some slight dullness of the intellect, some hebetude, or even a slight amount of melancholia. In certain cases the patients are subject to attacks of impulsive laughing; that is to say, without any sufficient cause they suddenly break out in exaggerated laughter, from which they quickly recover themselves. These attacks are due the cerebral lesions. As the patient gets worse the paraplegia increases and he becomes finally helpless. The use of the arms is preserved for a longer time. Involvement of the sphincter of the bladder occurs early, that of the rectum very late if at all.

The progress of the affection is variable. It usually progresses slowly with some remissions, reaching finally in two or three years a chronic stage, in which the patient remains for several years without much change. At other times the progress of the disease is hastened by repeated exacerbations, accompanied by apoplectiform or hemiplegic attacks.

In still other cases the amelioration continues and remains permanent, and a practical cure takes place.

Types.—The various symptom groups of the disease are classified as cerebro-cerebellar, and spinal. The cerebro-cerebellar symptoms consist in modifications of speech, attacks of vertigo and apoplectiform seizures, hemiplegia, intention tremor, labyrinthine symptoms, mental changes, optic atrophy and spasmodic laughing or crying. There is sometimes also a certain amount of deafness and perversion of taste and smell.

In the not uncommon spinal type, the dominant symptoms are the spasmodic paraplegia, with some bladder and sexual weakness, and a slight amount of sensory trouble. There may be also some weakness and unsteadiness of the arms, but the cranial nerves are not involved.

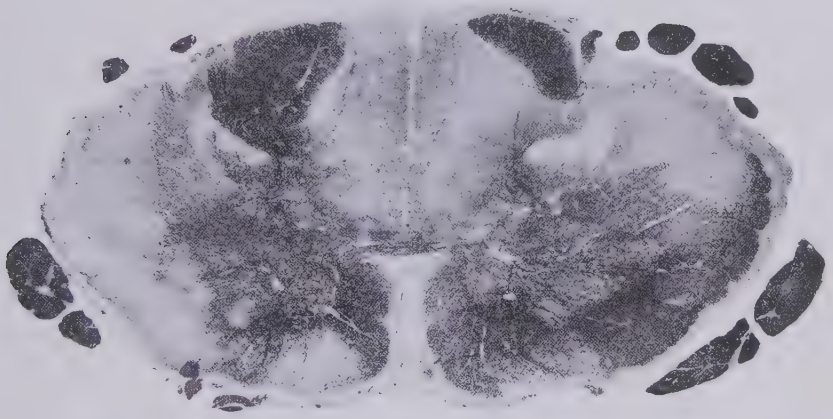


FIG. 207.—Multiple sclerosis, showing lesions of the cord. (*From photograph by Dr. Charles I. Lambert.*)

Aborted Types.—In some cases the nodules of sclerosis are so limited in number and so peculiarly placed that they give rise to very atypical and mild forms of the disease.

Pathology.—Grayish nodules are found distributed through the brain and spinal cord (Figs. 207–208). They vary in size from a millimetre to two or three centimetres in diameter (one twenty-fifth to one inch). They are of firmer consistence than is the surrounding brain substance, but are not quite so hard as is ordinary connective tissue. They consist microscopically of neuroglia tissue, which some assert to be connected with the walls of the blood-vessels, as a rule. Very often the axis-cylinders of nerves can be seen passing through the lesion. The nodules are found most frequently in the white matter of the brain, more especially in the pons, internal capsule, and centrum ovale. They rarely

begin primarily in the gray matter, but may invade it secondarily. The roots of the peripheral, especially of the cranial, nerves occasionally contain or are surrounded by these sclerotic masses. In the spinal cord they may extend up and down the gray and white matter for a considerable distance, or they may involve the whole cord at a certain level, turning it into a fibrous mass. The blood-vessels surrounding and in connection with these diseased areas show some evidences of thickening and increased vascularity, but no true inflammatory process, as a rule. The primary pathological change in multiple sclerosis is as yet unknown; many things point to its starting originally from small emboli or thrombi which lead to minute softenings, with a secondary reparative and sclerotic

Fig. 208.—Multiple cerebrospinal sclerosis. (*Charcot.*)

process. The fact that the disease follows infective fevers makes such an origin of it seem probable. On the other hand, pathological anatomy does not yet bear out this view, and it is possible that the disease begins by a primary degeneration affecting first the myelin sheaths of the nerve-fibres sparing the axis-cylinders and cell bodies, this being followed by a neuroglia and connective-tissue proliferation which ends in the formation of the small islands of sclerosis. An important pathological peculiarity of the process is that, while it destroys the myelin sheaths of the nerves, the axis-cylinders remain intact for a long time, and con-

sequently conduction of nerve impulses takes place imperfectly, directly through the nodular masses.

Course and Duration.—The disease runs a very irregular course. Its prodromal stage is long and remissions of considerable length occur. The disease may last from ten to thirty years, the average duration being five to ten years. Death sometimes occurs from involvement of the nerves of the medulla, but more often from weakness and exhaustion or some intercurrent malady.

Diagnosis.—The diagnosis in typical cases is not very difficult; but as, on the other hand, typical cases are not common, the disease has always to be studied with great care before certainty can be reached. The diagnosis is based upon the slow development of the disease, with attacks of vertigo, weakness, paræsthesia and uncertainty in gait; also upon the paralysis of the extremities, the intention tremor, ataxia, rigidity and contractures; upon the disturbances of vision, nystagmus; loss of abdominal reflexes, and the speech troubles. The existence of early paralysis with paræsthesiæ, or of paræsthesiæ alone followed by remission, is most significant. The presence of headaches, attacks of vertigo, apoplectiform attacks, and the peculiar mental condition often furnish help. The age of the patient and the cause should also be taken into consideration. The disease must be distinguished from Friedreich's ataxia, spastic spinal paralysis, locomotor ataxia, dementia paralytica, bulbar paralysis, paralysis agitans, chronic meningitis, and hysteria. The points already given and those furnished under the heads of these different diseases must be utilized in making these distinctions. In the pseudo-sclerosis of Westphal, there are no ocular symptoms. In diffuse cerebral sclerosis, also, ocular symptoms are rare and there are progressive dementia and paralysis. In multiple sclerosis the pupils are rarely affected. The method of exclusion may be used with advantage in reaching the diagnosis of this protean malady.

Prognosis.—The prognosis, while not favorable as regards the ultimate cure, is somewhat favorable as regards a remission and improvement, and the disease on the whole is not so severe as is locomotor ataxia or the other degenerative disorders.

A quiet regular life is very necessary, and at certain intervals the patient *should go to bed for several days.* Systematic re-educational exercises are to be used for very short periods. Electricity is of little value except for symptoms. Hypodermic injections of the cacodylates; also those of salvarsan and of fibrolysin are useful. Urotropin may be given for short periods in large doses. Occasional courses of iodides and mercury are beneficial. Atropin and belladonna are useful symptomatically.

If the case is taken early and the patient works for his cure hopefully and earnestly much may be accomplished.

THE APOPLEXIES

Apoplexy is a clinical term used to indicate a condition characterized by sudden paralysis, usually attended with loss of consciousness, and due to the breaking or blocking up of a blood-vessel in the brain.

Apoplexy is a general term. Particular forms are described in accordance with the cause of the apoplexy. These are:

1. Intracranial hemorrhage, from rupture of a blood-vessel (hemorrhagic apoplexy).
2. Acute cerebral softening, from embolism or thrombosis (embolic or thrombotic apoplexy).

APOPLEXY FROM INTRA-CRANIAL HEMORRHAGE

(Cerebral Hemorrhage, Hemiplegia)

There are four groups of intra-cranial blood-vessels: those of the dura mater, those of the pia mater, those supplying the basal ganglia and white matter, and those supplying the pons, medulla, and cerebellum. These last are chiefly branches of the vertebrals and are a separate group, subject to somewhat different mechanical conditions. Corresponding to this we have:

1. Dural or pachymeningeal hemorrhages.
2. Pial or subarachnoid hemorrhages.
3. *Central or capsular hemorrhages.*
4. Hemorrhages in the medulla, pons and cerebellum.

It is the central hemorrhages (No. 3), due to rupture of the blood-vessels going to the great basal ganglia, internal capsule, and white matter, that constitute the great majority of cerebral hemorrhages seen by the physician. It is this class that I have particularly in mind in the following description.

Etiology.—At the time of birth and during infancy there is a slight tendency to intra-cranial hemorrhage owing to the accidents and injuries of labor. The hemorrhages at birth are usually meningeal and traumatic. After this period the liability is very small, but slowly increases up to the age of forty, when predisposition specially begins. Four-fifths of all cases occur after forty, and the tendency to hemorrhages increases in each decade up to eighty, when it diminishes absolutely and relatively. Males are slightly more predisposed than females. Rather more cases occur in cold weather, at high altitudes, in the temperate zone, and among civilized races. With the improvement in general health conditions and the greater average longevity, there is an increase in apoplexies. Hereditary tendencies are most important. This tendency is to arterial degeneration and often runs in families. Excessive eating and drinking,

excessive work, and anxiety of mind, in fine all the causes leading to arterial disease, predispose to cerebral hemorrhage. Syphilis is more often a cause of thrombosis. It was present in over 20 per cent. of my cases of cerebral hemorrhage, while kidney disease was present in one-third of the cases.

Congenital anomalies, such as a narrow thoracic aorta or inherited deficiency in the strength of the walls of the blood-vessels, also play a part. Any sudden physical exertion, such as straining at stool, the excitement at coitus or of a passion, eating a large meal and drinking a great deal of fluid, especially alcohol, taking a cold bath, all may lead to rupture of an artery. Direct blows on the head may cause meningeal hemorrhage, rarely a central hemorrhage, for the central blood-vessels are well protected.[1]

The symptoms are the prodromal, those of the attack and acute stage, and those of the chronic stage.

Prodromal symptoms are rare except in syphilitic cases. When present the patient suffers from headache, insomnia, dizziness, numbness of the hand and foot on one side, and a failure of memory for words. He may have "full" feelings or even pain in the head and bad dreams at night. Nosebleed and irregular heart action sometimes occur. Often attack comes on when the patient is feeling particularly well. The attack always comes on suddenly and may be accompanied (1) by convulsions and coma, (2) by coma alone, or (3) it may come without loss of consciousness.

1. Initial convulsions are rare and generally mean a meningeal hemorrhage. They occur, however, in young children. When present they are unilateral or partial, as a rule, but may be general. 2. The common mode of onset is with coma. The patient, without warning, suddenly becomes dizzy, loses consciousness, and falls. The face is flushed, the pulse hard and rather slow, the blood-pressure may rise to over 200 mm., the breathing is labored and stertorous, the cheek on one side puffs out with each expiration, the eyes are partly closed, the eye-balls fixed or deviated to the paralyzed side, the pupils are contracted and rigid, the skin is bathed in sweat, the limbs are relaxed, but some evidence of hemiplegia is present and the Babinski reflex can be usually elicited; the urine may be retained or it and the feces involuntarily evacuated. The urine is usually of rather high specific gravity and often contains albumin, even when there is no renal disease. The temperature in severe cases may fall below normal during the first twelve hours, even to 96° F., but this is not the rule. It is the rule, however, for the tem-

[1] Among 324 cases of hemiplegia occurring in the Cornell clinic, carefully analyzed by Dr. T. W. Evans, he found less than 1 per cent. in which even the question of trauma could be raised. Among 120 private cases, there was a history of a blow and of syphilis in one.

perature in a few hours to be ½° or 1° higher on the paralyzed than on the sound side. If the case is rapidly fatal, coma continues, respiration often assumes a Cheyne-Stokes character, the pulse becomes fast, the blood tension falls and symptoms of bulbar failure occur; the temperature gradually rises, and usually reaches 102° or 103° F., until just before death, when it may sink again. Swallowing and speech become difficult, hypostatic pneumonia sets in, and the patient dies in from two to four days. In slower fatal cases the patient regains consciousness partially and then enters a condition of stupor or mild delirium. He is restless and suffers from headache. The temperature may continue normal for a time, but is usually higher on the affected side. At the end of two or three weeks it rises higher, pneumonia may develop, the patient becomes unconscious, and death ensues. In the favorable cases, which constitute the majority, coma, if present, gradually passes away in from one to six hours, leaving the patient's mind somewhat weak and confused and his speech disturbed, or more rarely the intelligence may not be at all disturbed. During the first few days or weeks after the attack the physician finds that the prominent symptom is the hemiplegia. This affects the arm and leg most and the face least. Only the lower two branches of the facial nerve are involved, and the patient can shut the eyes. The tongue, if protruded, turns to the paralyzed side; the uvula is turned in various ways and its position is of no significance. There is often some evidence of cutaneous anæsthesia of the paralyzed side, and less often hemianopsia and disturbances of hearing occur. In right-sided hemiplegia the patient, after recovering consciousness, is often unable to talk or to understand what is said. Examination shows that he has a motor or sensory aphasia (*vide* Aphasia).

Fig. 209.—Chronic hemiplegia with contractures. (*Curschmann.*)

The deviation of the eyes and head to one side usually disappears in a day or two. Occasionally there is a temporary ptosis. The pupils at first are contracted, that on the paralyzed side the more so; this condition disappears with returning consciousness.

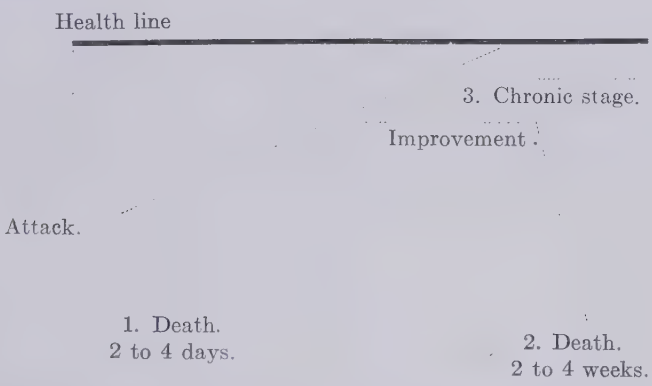
The paralysis of the arm and leg is usually flaccid at first, and the limb falls heavily when lifted; the reflexes are lessened or abolished. In a few days rigidity and exaggeration of the deep reflexes appear on the paralyzed side, with ankle clonus and dorsal flexion of the big toe.

Sometimes, however, rigidity sets in at once. This symptom occurs when the blood has broken into the ventricles, and also in some meningeal hemorrhages. The skin reflexes, particularly the abdominals are lost on the paralyzed side.

The usual course of the temperature is for it to rise on the second and third day to 100°F. or 102°F., being ½° to 1° higher on the paralyzed side. In a few days it gradually falls, so that by the eighth to the tenth day it is normal.

If the temperature continues to rise after the fourth or fifth day, it is a sign of an extension or inflammatory reaction of the hemorrhage. Hence the thermometer furnishes a very important criterion of the seriousness of the case.

The varying course of the apoplexy is shown in the following diagram:



The Chronic Stage, Hemiplegia.—The description of this condition applies almost equally to all forms of apoplexy with hemiplegia. At the end of a month, if fever and symptoms of cerebral irritation have subsided, the chronic stage may be said to begin. The hemiplegia has improved, the patient can move the leg and arm a little, sensory symptoms have lessened, the mind is clear, headache has disappeared. Improvement continues, though more slowly, for several months or even one or two years. During this time the patient is "a hemiplegic."

The hemiplegia affects the arm more than the leg, and the face least

of all. The distal segments of the limbs, the feet and hands, are affected more than those near the trunk. The muscles that act bilaterally, such as those of respiration, phonation, and facial expression, are but slightly involved. The paralysis is not strictly a hemiplegia, for the muscles on the sound side are somewhat weakened, as tests will show. In severe cases, especially in old people, even the visceral muscles, especially those of the bladder, are weakened. At the onset of the attack there is sometimes a temporary "initial" rigidity of the muscles on the paralyzed side, or an "early" rigidity may develop in one or two days. There always develops at about the beginning of the second week a "late" rigidity. This, which at first is slight, gradually increases, and finally contractures affect the paralyzed limbs. The superficial reflexes, which at first were absent rarely reappear; the tendon reflexes

FIG. 210.—Showing athetoid movements of hands. (*Curschmann.*)

become much exaggerated, and clonus can be obtained in the leg and arm. The sound side shares to a small extent in these conditions. The contractures affect the extensors of the foot more than the flexors, and bring the toe down and the heel up. The leg is held nearly extended, and the limb in walking is swung around, the toe scraping the ground. The shoulder is adducted, the forearm flexed, and the fingers are tightly shut into the palm by the overaction of the flexors (Fig. 209). The facial muscles show a slight contraction and drawing to the affected side. The muscles on the paralyzed side do not waste. In infantile hemiplegia, however, the affected limbs grow less than those on the sound side.

The paralyzed limbs may be the seat of peculiar disorders of movement. These consist of:

Associated movements, tremor, ataxia, choreic movements, continu-

ous or athetoid movements (Fig. 210), spastic movements and cramps. Such movements, aside from those that are spastic, are rarely seen in the hemiplegia of adults.

The electrical irritability may be at first slightly increased or diminished, but the change is small in amount and never reaches the degenerative stage. The direct muscular irritability is at first increased on the affected side.

Hemianæsthesia, if present at first, disappears to a great extent, leaving only residua about the feet and hands. Paræsthesiæ are common. In rare cases the patient suffers great pain in the arm and leg. This pain is generally of a neuralgic or burning character and very obstinate and distressing. Cramping pains in the legs and arms are common in the severer cases.

During the first few weeks after the onset, the temperature of the hemiplegic side is usually a very little higher than that of the sound side.

Vasomotor disturbances, sweating, skin eruptions, and increased growth of hair are some of the rarer symptoms.

The mental condition is sometimes affected. The patient becomes irritable, cries easily, and is in general more emotional. The memory is impaired, and the power of concentrating the attention and carrying on work is less. Sometimes a progressive mental deterioration sets in and epilepsy or insanity develops. The mental disturbance is greater in old people and depends somewhat on the size of the hemorrhage. Those forms which produce serious aphasia especially limit and lessen mental activity.

Dural Hemorrhages.—Hemorrhages from the vessels of the dura mater are usually due to a rupture of the middle meningeal artery or vein or some of the cerebral veins as they pass to the sinus, and this is especially true in such hemorrhages as are the result of injuries to the head. The causes are blows on the head, obstetrical injuries, alcoholism, and the dementias.

In dural hemorrhages the *result of blows on the head*, the clot is sometimes intra-dural, lying in the arachnoid space, and sometimes epidural, lying between the bone and the dural membrane. The extra-dural hemorrhages are perhaps a little more common in surgical experience; the intra-dural in medical and especially asylum experience. In over one-half of extra-dural cases there is an interval of consciousness lasting from a few hours to two months, but usually only a few hours, between the accident and the time when distinctive cerebral symptoms develop. Then the patient gradually becomes dull, somnolent, and finally comatose. Along with the gradual or rather sudden loss of consciousness there develops a hemiplegia upon the side opposite the clot. This is usually not complete, though it may become so. It is much less

common in extra-dural hemorrhage. Anæsthesia is rarely present. The reflexes are generally somewhat exaggerated, and there may be considerable rigidity. Spasmodic movements of some kind occur in nearly half the extra-dural cases and in more than half of the intra-dural. These spasmodic movements may involve the whole of the affected side, or may simply affect the eyes and the facial muscles. They consist of irregular twitchings. The pupils are usually somewhat contracted, more so upon the paralyzed side. When there is a dilated pupil on the side of the lesion and a small pupil on the opposite side, it is known as the *Hutchinson pupil*, and means a severe brain compression involving the third nerve at the base. The eyes are generally both turned toward the affected side and away from the lesion. The pulse is hard, slow and full; the tension high; the respiration is rarely stertorous, though it may sometimes be so, and Cheyne-Stokes respiration may be present. In these cases the clot is very large and the compression great. Aphasia may be present if the clot is upon the left side. The temperature may be raised one or two degrees, or it may be normal. The progress of the disease is usually steadily fatal unless surgical interference is undertaken. The coma deepens, the respiration becomes stertorous and then embarrassed, the pulse gets rapid and weak, and the patient dies. With surgical interference (since 1886), between two-third and three-fourths of the cases are saved (Scudder and Lund).

Subdural hemorrhages not caused by injuries are due sometimes to the rupture of a meningeal artery, but usually to rupture of the veins of the pia mater or to the rupture of new vessels formed in a pachymeningeal exudate. This spontaneous hemorrhage is rare in ordinary practice, but is not specially so in insane asylums or in large city hospitals. This is bebecause the two great causes of this type of hemorrhage are the dementias and alcoholism. General paresis is the form of insanity with which it is oftenest associated. In the case of alcoholics, it is probable that injuries from blows are an exciting factor in the production of the hemorrhage, these occurring while the patient is in a state of intoxication. The symptoms of these hemorrhages are extremely variable, owing to the complicating influences of the insanity and alcohol. The patient, after suffering from headaches or vertigo, becomes suddenly comatose and shows marked evidences of hemiplegia and even of hemianæsthesia. Rigidity of the paralyzed side is often present, and sometimes spasmodic movements are observed. On the other hand, at times the paralysis can hardly be observed, and the patient is in a semicomatose state, has a muttering delirium, and presents the general aspect of a person suffering from the œdema or "wet-brain" of alcoholics. In dural hemorrhages occurring in paresis, the patient usually without warning becomes unconscious, and he often has some convulsive symptom and a hemiplegia develops. In

these cases there is often a rapid improvement, and the patient gets partly well, usually experiencing other attacks later.

Pial Hemorrhages.—Hemorrhages from the pial arteries are rare and of small extent.

The most frequent cause is trauma, associated perhaps with syphilis and alcoholism. In many instances very slight localizing symptoms occur, and no absolute diagnosis can be made. If the hemorrhage, however, is in the motor area of the cortex, local spasmodic movements and some hemiplegia are observed. The most characteristic symptoms are the sudden incomplete hemiplegia, involving, perhaps, mainly an arm or a leg, associated with local spasmodic movements, resembling Jacksonian epilepsy.

Hemorrhages due to rupture of large pial veins occur most often as obstetrical injuries. The hemorrhages here may cause convulsions, paralysis and death, or lead to permanent mental and physical defects.

Hemorrhages of the Vertebral Artery and its Branches.—*Hemorrhage in the medulla* is extremely rare. If of any size, it causes instant death; if small, it will leave symptoms similar to those of softening.

Hemorrhage of the pons is also rare, but it makes up 2 per cent. of my cases. The cause is much the same as that for cerebral hemorrhages, though syphilis is less often a factor. In nearly half the cases the hemorrhage is a large and immediately fatal one. In the majority of cases it is a small focal hemorrhage from rupture of a transverse or median branch of the basilar artery.

The patient has some prodromal headaches and malaise for a few days, with vertigo and sometimes vomiting. Then he falls suddenly as if by a lightning stroke, into a coma, usually very profound. There are twitchings of the face or of the limbs or both, but rarely any general convulsion (three cases). The face is flushed, there is a fall of temperature and the pulse is irregular, but not markedly increased or slowed; as a rule, the respiration is slow, 4 or 6 per minute, or more often irregular, and of Cheyne-Stokes type. The pupils are contracted to a pin-point, and do not respond to light, but may be uneven. There is convergent strabismus or conjugate deviation of the eyes. The limbs are at first stiff, but may be relaxed later and the reflexes increased.

The patient cannot be aroused, but can be made to vomit. A few hours later the temperature rises, sometimes very high—106 to 108—but usually not. There are involuntary movements of the bowels, swallowing is impossible; evidences of dyspnœa are shown. The pulse becomes more rapid, cyanosis develops, the patient dies in six to twenty hours, usually with evidence of paralysis of respiration.

If the hemorrhage is less severe, the coma is less profound or may

be absent, the patient shows signs of agonizing suffering and difficulty in respiration, evidences of crossed paralysis, crossed anæsthesia; and disturbance of equilibrium and ataxia can be noted.

In still smaller lesions we find no such severe general symptoms, but only those due to a circumscribed focus in one side of the pons, because the smaller lesions are unilateral. The syndrome of serious pons lesions then is:

1. Headache, malaise, vomiting.
2. Sudden and profound coma.
3. Twitching of the face and limbs or both.
4. Myosis and convergent strabismus or conjugate deviation (away from the side of the lesion).
5. Slow, irregular breathing.
6. Irregular pulse.
7. Dysphagia.
8. Paralysis of limbs or crossed paralysis and exaggerated reflexes.
9. Gradual rise of temperature, sometimes to high point.
10. Death inside of twenty-four hours.

Cerebellar Apoplexy.—Hemorrhage into the cerebellum occurs in 1 or 2 per cent. of all fatal cases. Its recognition is very difficult. There is sometimes a preliminary period of severe headache, lasting several days. In other cases the patient at once falls into a state of profound coma, with stertorous respiration. Vomiting sometimes occurs. There may be some hemiplegia, and if so this is on the side of the lesion, owing to this pressure on the motor tract. Distinct evidences of hemiplegia, however, are not always observed. The condition of the pulse and arterial system is very much like that of ordinary apoplexy, but the respiratory system is usually more seriously affected. Disturbances in the movements of the eyes and in swallowing, and in fact all those symptoms which show a pressure or irritation due to blood oozing into the fourth ventricle may be present. Death is almost sure to occur, and is inevitable if the hemorrhage, as is so often the case, breaks through and reaches the fourth ventricle.

If the hemorrhage is small and does not break into the ventricle, the patient suffers from intense vertigo which obliges him to lie perfectly still in a horizontal position. With or preceding this he may fall down and have convulsive movements or forced and irregular tonic contractions of the muscles; in other words, a cerebellar fit. If this passes away the vertigo also may pass, and the patient gets nearly well, but is left with some feeling of uncertainty in gait, and with tinnitus cerebri and tendency to vertiginous and cerebellar attacks.

Pathology and Morbid Anatomy.—Spontaneous intra-cranial hemorrhage is always due to the presence of diseased blood-vessels in the brain. This diseased condition consists of:

1. A degenerative arteritis which results sometimes in producing small aneurisms. 2. A fatty degeneration of the vessel walls. 3. Besides this, in most cases the larger blood-vessels are atheromatous.

The arteritis may produce small or miliary aneurisms which involve only the smaller arteries, especially those of the central group. They may be fusiform or sacculated in shape; they range in size from one-fourth to one millimetre ($\frac{1}{100}$ to $\frac{1}{25}$ in.) in diameter. They are usually not very numerous, but there may be as many as a hundred in the brain. They are the results, not of inflammation, but of a degeneration which affects first an area in the internal coat; this causes local weakness and consequent dilatation; secondarily there is a peri-arteritis. These aneurisms occur almost exclusively during the degenerative period of life.

Fatty degeneration of the walls of the small cerebral arteries occurs in purpura, scurvy, marasmic conditions, and post-infective states, especially in early life, and is the common cause of hemorrhage at that time.

Atheroma affects the larger vessels only. It is indirectly a cause of hemorrhage by lessening the elasticity of the vessel wall. Atheroma is present in from one-eighth to one-fifth of all cases. Hypertrophy of the heart is a factor in causing hemorrhage, and such hypertrophy exists in about 40 per cent. of cases. Emboli lodged in the cerebral arteries may cause hemorrhage by suddenly stopping the arterial circulation and raising the blood-pressure. Hemorrhages are found by far the oftenest (20 per cent.) in the caudate and lenticular nuclei and adjacent parts. The lenticular and lenticulo-striate branches of the middle cerebral are oftenest affected; next the branches of the anterior cerebral to the caudate nucleus and the lenticulo-optic branches of the middle cerebral. The branches of the posterior cerebral break more rarely. The parts affected in hemorrhage in fatal hospital cases, in order of frequency, are about as follows (Hoobler).

Frontal lobe	24
Parietal lobe	14
Occipital lobe	6
Temporal lobe	14
Corpus striatum	62
Optic thalamus	25
Internal capsule	28
Ventricles	64
Corpus callosum	1
Corpora quadrigemina	1
Piæ and cortical	8
Pachymeningeal	7
Pons	1
Cerebellum	3
	258

These figures show that over three-fourths of all the hemorrhages occur from rupture of the central or ganglionic arteries.

Cortex hemorrhages are generally small and may be subarachnoid or may break through into the arachnoid cavity. Ventricular hemorrhages are almost always secondary to a rupture into the neighborhood of the basal ganglia. Pons hemorrhages occur usually in the median line. Cerebellar hemorrhages are oftenest due to rupture of the superior cerebellar artery. They usually cleave their way externally and break into the fourth ventricle. Dural hemorrhages are due to rupture of the meningeal veins and arteries and of the vessels in newly organized clots. They lie in the arachnoid cavity and flatten the convolutions.

The reparative changes after a hemorrhage take the following course: 1st. Coagulation of the blood, which in a few days begins to soften and become absorbed. 2d. Formation of a fibrinous wall about the clot. This occurs from the seventh to the ninth day. 3d. Formation of a cyst with transparent fluid contents, and perhaps fibrous trabeculæ running through it, twentieth to thirtieth day. 4th. Contraction of the cyst wall, which begins by the fortieth day. 5th. Secondary degenerations begin from the tenth to the fourteenth day. These degenerations vary with the location of the lesion. The most important one is that of the pyramidal tract. This is more or less completely sclerosed from the lesion to the spinal cord.

The diagnosis of hemorrhagic apoplexy must be made from alcoholic coma, uræmic coma, diabetic, opium and other types of coma, epilepsy and hysteria; acute softening from embolism and thrombosis.

From alcoholic coma the diagnosis is made by the odor of the breath, the incomplete coma, the equal pupils, the absence of low or unequal temperature, the absence of paralysis, the equality of the reflexes.

From uræmic coma by the presence of albumin and casts in the urine, though their presence does not surely indicate uræmia; by the equal pupils, the temperature, the absence of hemiplegia and the presence of the physiognomy peculiar to cases of chronic Bright's disease.

From diabetic coma by the odor of the breath and the condition of the urine.

From opium poisoning by the history, the stomach contents, the presence of equal and contracted pupils, the slow respirations, the temperature, and the absence of paralysis.

From epilepsy by the history of the onset with epileptic cry, the dilated and equal pupils, the biting of the tongue, the absence of hemiplegia, the rather rapid return of consciousness.

Hysterical attacks present little semblance to that of apoplexy; consciousness is not profoundly lost; the deep and superficial reflexes are not greatly changed; the hemiplegia is characterized by its flaccidity,

by its not involving the face, and by the presence of the anæsthesias and other hysterical stigmata.[1]

In embolic softening the earlier age of the patient, the presence of decided valvular heart disease, the parturient condition, the slighter degree and shorter duration of coma, the absence of serious disturbance of temperature, the onset first of paralysis and then of convulsive movements and coma—all lead to a presumption in favor of embolism.

The presence, on the other hand, of a congested face, tense pulse, and throbbing carotids favors the existence of a hemorrhage.

From thrombotic softening diagnosis is more difficult. The existence of syphilis is in favor of thrombosis. The occurrence of prodromata, consisting of slight seizures quickly recovered from, the slighter degree of coma, the advanced age, hard atheromatous arteries, low blood-pressure, evidence of anæmia and asthenia, weak or fatty heart, the absence of stertorous respiration, flushed face, and unequal temperature not much lowered or raised, the slight pupillary disturbance, and absence of convulsions point to thrombotic softening. Evidence of a lesion in the pons or cerebellum unless there is syphilis suggests hemorrhage, while evidence of lesion in the medulla points almost surely to softening. The existence of a very high blood-pressure will often help the diagnosis in all cases, but especially from softenings.

Several authors have found large granular macrophages containing red blood-cells in the cerebrospinal fluid in cerebral hemorrhage, but these are not always found (Hastings). The examination of the urine for hæmoglobin has also been used as a diagnostic test.

Lumbar puncture and the finding of blood in the cerebro-spinal fluid indicate a ventricular or surface hemorrhage.

The chances in any case between the ages of thirty and fifty, in a city hospital practice, if there is no heart disease, are six to one in favor of hemorrhage, if syphilis can be excluded.

The diagnosis of the location of the lesion has already been indicated, except for the central hemorrhages. These represent four clinical types:

1. That due to rupture of the lenticulo-striate branch of the middle cerebral with injury of the lenticular region and internal capsule in its more anterior part. This gives the picture of the usual type of hemiplegia as described with no marked sensory symptoms.

2. If the lenticulo-optic is involved, there is less hemiplegia and the leg is more involved, and there is involvement of sensation.

[1] Hoover's sign of hemiplegia: The patient lies on his back. The examiner places his hand under the patient's heel on the side of the paralysis and directs him to lift the sound leg. In an organic hemiplegia the examiner feels that the patient's heel presses down against his hand, when the patient lifts the sound leg. This does not occur in hysterical hemiplegia, nor upon lifting the paretic leg, nor in the normal subject.

3. If the thalamic arteries are involved, there is a temporary hemiplegia with appearance of the thalamic syndrome.

4. Ventricular hemorrhages are usually the result of very extensive lenticulo-striate lesions, the blood finally bursting into a lateral ventricle and thence permeating into all the ventricles and even to the subarachnoid space, so that a diagnosis can be made or confirmed by a lumbar puncture. The symptoms are characterized by the severity of the coma, giving way of the medullary centres, a motor restlessness, increased rigidity of the limbs, and sometimes evidence of bilateral paralysis. The contracted pupils, loss of power over the sphincters, sweating, vasomotor disturbances, rise of temperature, irregular respirations, and

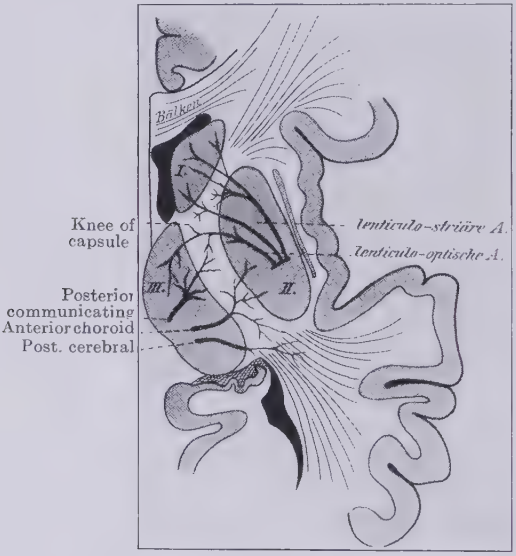


Fig. 211.—Blood-supply of basal ganglia and internal capsule. (*Bing.*)

rapid pulse indicate a serious cerebral lesion. The blood-pressure, which is high when there is a large clot on the brain substance, falls if it breaks into the ventricles.

Prognosis.—The majority of cases apparently get over the first attack of apoplexy, but it is possible that many of these cases are thrombotic rather than hemorrhagic. My view, however, is that since the majority of hospital cases recover and since the majority of these are hemorrhagic there is a large number of recoveries. They are very liable to have another within one to five years. The minority recover from this. Few survive a third attack. The prognosis of the attack itself depends on the severity of the coma and paralysis, the disturbance of temperature and of

respiration, the evidence of rupture into the ventricles, the development of decubitus, the continuance of loss of control over the bowels and bladder.

If profound coma continues four days there is little hope; if fever develops and continues steadily, or if there is initial subnormal temperature, or if signs of bulbar compression and paralysis occur, the progonosis is grave.

If the patient passes the first week with little or no fever and consciousness has returned, the prognosis is good.

The presence of renal disease and of alcoholism is bad. Development of slight delirium which continues is unfavorable.

Cerebellar and pons hemorrhages are very fatal, meningeal less so.

The prognosis of the chronic stage has been given under symptoms.

Improvement continues rather rapidly for three months, then very slowly. Improvement may continue for one or two years. Complete recovery is very rare. The great danger after middle age is recurrence of the attack.

Something of the course of the apoplexies in a large city hospital is shown by the following statistics:

In three recent years there were admitted to Bellevue Hospital 315 cases of cerebral apoplexy with hemiplegia. Of these, 115 died and of these 26 were autopsied. In 10 cases the hemorrhage involved the central arteries and broke into the lateral ventricles. In 16 the lenticular region and capsule only were involved.

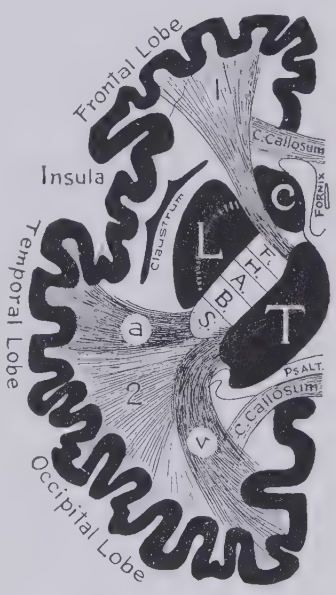


FIG. 212.—Diagrammatic representation of the internal capsule and the cortical tracts that pass through it. 1, 2, Fronto-occipito-pontine efferent; *a*, *v*, auditory, visual, afferent; *F*, cortico-spinal from face center; *H*, hand; *A*, arm; *B*, leg; *S*, sensory tract. (*Bing.*) *C*, *T*, *L*, and the internal capsule are the parts most often involved in hemorrhage.

Treatment of the Attack.—The patient should be laid in a horizontal position and kept quiet. Ice may be applied to the head and hot bottles at the feet, only if these measures seem indicated. They usually do no good. A laxative should be given, either one or two drops of croton oil or a quarter of a grain of elaterium. If there is evidence of intense cerebral congestion, the pulse being very full and hard and the heart beating strongly and blood-pressure high, bleeding eight to twelve ounces is justifiable. Ordinarily it is better to give tincture of aconite every twenty minutes for two or three hours.

Administration of bromide of sodium and enemata of ergot have been advised, but are of doubtful value. After the first twelve hours, treatment must be symptomatic. Should delirium and other evidence of mental irritation appear, chloral hydrate or morphine and hyoscin are indicated, and an elaterium purge, given if the patient is not too weak. The use of iodide of potassium or mercury is not indicated unless the case is distinctly syphilitic. Great care should now be taken that the patient does not develop pneumonia. The mouth and pharynx should be cleansed antiseptically, and the patient should not be allowed to remain in one position. If there is sufficient evidence of a meningeal or cortical clot, trephining should be seriously considered.

At the end of three or four weeks or even earlier careful massage should be begun and continued daily. With it the faradic battery may be used on the affected limbs. When contractures develop the stabile galvanic current may be tried, though it does little good. Static sparks, however, are helpful; lukewarm baths should be tried and measures used to produce a hyperextension of the affected parts.

Internally during this time the patient is to be given courses of iodide of potassium, tonics, and laxatives if needed. The patient should be made to live a quiet life, preferably in a warm, equable climate. The kidneys should be kept active and arterial tension only moderately low. For these purposes nitroglycerine should be given and at times small doses of chloral, and the diet should be simple and rather non-nitrogenous. Strychnine in very small doses (gr. $\frac{1}{100}$) sometimes helps the contractures, as also do the bromides and physostigma.

APOPLEXY FROM ACUTE SOFTENING

(Embolism, Thrombosis)

Acute softening is a condition caused by the plugging of a blood-vessel with an embolus or thrombus, and is characterized by a comparatively sudden (embolism), or gradual (thrombus), apoplectic seizure, the symptoms eventually running a course much like that of cerebral hemorrhage.

Etiology.—Embolism occurs rather more often in women, thrombosis in men. Embolism is rare in children; it occurs oftenest between the ages of twenty and fifty, thrombosis between the ages of fifty and seventy. The most important predisposing factors in embolism are acute or recurrent endocarditis, infectious fevers, profound anæmia, pregnancy, and blood dyscrasias; in thrombosis, arterial sclerosis from any cause. Severe gas poisoning leads sometimes to thromboses. The same causes which lead to the arterial disease which produces cerebral hemorrhage also predispose to thrombosis, though in the latter condition atheroma and obliterating arteritis play the important part.

Symptoms.—In embolism there are rarely any premonitory symptoms; the onset is sudden; it may begin with some convulsive twitchings, then follow hemiplegia and temporary loss of consciousness. Coma, however, is rarer than in hemorrhage, and if present is usually shorter. There is rarely vomiting, nor do we find the hard, pulsating arteries, flushed face, and severely stertorous breathing. The initial temperature changes are slight, but in a few days fever may develop.

In thrombosis premonitory symptoms are frequent. In syphilitic cases there are headaches and cranial-nerve palsies. In these and other cases vertigo, temporary aphasia, insomnia, transient hemiplegia, numbness of the hand and foot, and drowsiness may be present. The onset is more gradual; the hemiplegia slowly develops, taking several hours, or days and coming on with remissions, finally the patient gradually becomes completely hemiplegic often without coma. The attack sometimes is rather sudden, with no loss of consciousness, and it may occur in sleep. The temperature often has a slight initial fall, followed by a rise, just as in hemorrhage. In both embolism and thrombosis the hemiplegia tends to improve very much in a few days or weeks unless the vessel obliterated is a large one. Embolism is rather more apt to affect the left side of the brain, though the difference is not great. The middle cerebrals are most frequently affected. Thromboses affect the vertebrals, basilar, and posterior cerebral arteries more often relatively than do hemorrhages; then the initial symptoms may not present the character of hemiplegia, but of a ponto-bulbar paralysis or sensory hemiplegia. Acute softening may kill within twenty-four hours, but, as a rule, the patient survives the onset, and if he dies it is not for several weeks. After the acute stage is over the patient passes into the chronic stage, which resembles in nearly all respects that of hemorrhage. After an acute softening, however, it is believed that there are more spastic symptoms and a greater tendency to mobile spasm. In embolism, owing to the youth and freedom from arterial disease, the mind is less affected; while in thrombosis the contrary is the case.

In acute softening from gas-poisoning, a branch of the anterior cerebral artery which supplies the caudate and part of the lenticular nuclei is particularly affected so that a symmetrical softening of both lenticular nuclei is almost a characteristic of this accident. This is accompanied with coma and vasomotor and trophic disturbances of the limbs at times. In less severe cases there are disturbances of speech, tremor, irregular jerky movements and a temporary paraplegia with involvement of the deep reflexes.

Intermittent Hemiplegia (*hemiplegia sine materia*). Not rarely there occur attacks of hemiplegia which last only a few hours or a day or two. There is often with them mental confusion and aphasia. These attacks

are seen oftenest in cerebral lues; occasionally in epilepsy and migraine; and in other conditions in which there are arterio-renal defects. It is supposed that in some cases the paralysis is due to a local spasm of the cerebral blood-vessels (*e.g.*, migraine); in other cases to the temporary activity of an obliterating arteritis (lues); or in other cases to a local œdema perhaps associated with a uræmia or other toxic irritation.

Pathology.—The embolic plug cuts off the blood-supply from a certain area of brain tissue. In twenty-four hours this begins to soften. If the area is in the cortex it becomes red (red softening); if in the white and less vascular part, it is usually white with a few red punctate spots. The red softening gradually becomes yellow (yellow softening). The dead tissue gradually is absorbed, leaving a cicatrix or cyst. If the embolus contains infective microbes, there may be a local encephalitis and abscess.

In thrombosis there are usually evidences of extensive atheroma or syphilitic arteritis. In those instances in which the thrombosis is caused by the blood state and a weak heart, little arterial change occurs. Atheroma affects chiefly the internal carotids and the large arteries at the base, viz., the middle, anterior, and posterior cerebrals and the basilar and vertebrals. The branch of the anterior cerebral supplying the caudate and part of the lenticular nucleus is called "the artery of cerebral thrombosis."

Among seventy-two cases of cerebral softening with autopsy collected by Dr. B. R. Hoobler from hospital records, the site of the lesion was as follows:

Frontal lobe	6
Parietal lobe	17
Occipital lobe	4
Temporal lobe	9
Caudate nucleus	5
Lenticular nucleus	16
Optic thalamus	6
Internal capsule	6
Corpus callosum	2
Pons medulla	1
	—
	72

This shows that about half (35) involve the central and half the cortical arteries. The posterior cerebral is involved in about one-third of the cases. The parietal lobe and the caudate nucleus are most often affected. Most of these cases are thrombotic. Embolism almost always affects the great basal ganglia or some cortical branch of the anterior and middle cerebrals.

The secondary changes after thrombosis resemble those after embolism; a thrombus, however, may lead to supplementary embolism

through breaking off of a clot, and both conditions may cause a complicating cerebral hemorrhage.

The Diagnosis.—The important points have been gone over under the head of hemorrhage.

Very high blood-pressure, evidence of marked intra-cranial pressure, very sudden onset with profound coma, respiratory embarrassment, paralysis and fatal termination point to hemorrhage. Increased blood-pressure and temperature on the paralyzed side suggest hemorrhage. Lumbar puncture and withdrawal of cerebrospinal fluid may give definite eivdence of hemorrhage, but will not exclude it.

Premonitory symptoms covering one or more days indicate softening. A preceding period of feeling unusually well precedes some acute and fatal hemorrhages.

Slow steady loss of consciousness ending in very deep coma indicates hemorrhage.

Recurrent mild apoplectic seizures are probably always thrombotic. Very slowly developing seizures, coming on over a period of several days are thrombotic.

Embolism is distinguished from thrombosis by the age, the presence of endocarditis, of the puerperium or septic fever, and by its sudden onset, with perhaps some convulsive movements.

The prognosis as regards the attack is somewhat better than in hemorrhage, as a rule. In embolism it is good as regards recurrence; in thrombosis, bad, unless the trouble be due to syphilis or some preventable cause like gas poisoning. The recovery from attacks is more complete in acute softening. After the chronic stage is reached, however, the prognosis is about the same in all forms.

The treatment of the attacks consists essentially in rest and such attention to the bowels, kidneys, and heart as may be indicated. In thrombosis it may be important to give heart stimulants and arterial depressants, and for this purpose I advise the use of alcohol and of digitalis, or strophanthus with nitroglycerin. Thrombosis is much associated with syphilis and treatment must be given accordingly. It is also often associated with weakened and senile conditions and here individual treatment involving tonic measures, and rest are indicated. The symptomatic treatment of the chronic stage is the same as in hemorrhage.

SPECIAL FORMS OF HEMORRHAGIC OR THROMBOTIC SOFTENING

Small or large foci of softening attack the brain in different parts causing many symptom groups. A description of these is given under the head of softenings, as this is the most frequent condition.

Pseudo-bulbar paralysis or *cerebral bulbar paralysis* is a condition

characterized by double hemiplegia with involvement of the bulbar centres, so that there is difficulty in speech, mastication and swallowing. It is caused by a double lesion usually a softening involving the motor paths between the cortex and the nuclei in the centers of the motor V and of the VII, IX, XII nerves. Very rarely a one-sided lesion may cause this condition. The condition may occur in the aged and is then due to lacunar degeneration and softening.

As one sees it in the clinic and hospital, pseudo-bulbar paralysis occurs oftenest as the result of a second hemiplegic attack affecting the sound side of the brain in an old hemiplegia. Occasionally it comes on suddenly and is caused by acute bilateral vascular lesions. The seat of the lesions is usually in the neighborhood of the lenticular nuclei, and they are so located that they involve the fibres from the lower third of the motor cortex where the centres for the throat, tongue and face lie. The consequence is that there is no great involvement of the arms or legs.

The condition is a rare one. The symptoms are much like those of bulbar paralysis, but the diagnosis is easily made by consideration of the history, the absence of muscular atrophy and fibrillary tremor, and the marked involvement of the pyramidal tracts. The disease tends to improve rather than progress, as in true bulbar palsy.

Thalamus Apoplexy.—A hemorrhage here causes usually a temporary hemiplegia, with anæsthesia, which latter symptom continues. There may be later dysthesiæ and pains of the extremities and perhaps athetoid or choreic movements. Sometimes there is hemianopsia; generally the deep reflexes are not exaggerated. A group of symptoms has been noted forming the thalamic syndrome. It consists of:

1. Hemianæsthesia.
2. Hemiataxia.
3. Involuntary and spontaneous movements.
4. Central pains.
5. Cutaneous hyperæsthesia of the affected side, with overaction to pleasurable stimuli.
6. Hemianopsia; visual hallucinations.

In some cases there is paralysis of emotional facial expression on the affected side, while voluntary control of the facial muscles is retained.

Mid-brain softenings are not very rare. They are generally luetic or arterio-sclerotic. Hemorrhage and tumors are less common. Many different groups of symptoms may result. The accompanying Fig. 213 from Edinger, in connection with Fig. 214, shows how the different "syndromes" result in accordance with the location of the lesion. (See also Tumors of the Brain.)

Peduncular hemiplegia is usually due to a tumor, but there may be a softening, from a luetic meningitis or thrombosis. The lesion here

causes a paralysis of the third nerve on the side of the lesion and hemiplegia of the opposite side (Weber's syndrome).

Softening of the Pons.—Softening in the pons is most apt to be in the ventral part, near the raphe and next to this in the dorsal part of the pons. The basilar artery or its transverse branches are involved. Lesions of the pons alone are rarer than those involving also the medulla, especially its upper part, because the basilar is not plugged as often as the vertebrals and its branches. The prodromal symptoms are not so long. In a typical attack due to plugging of the basilar, we get a softening of the deeper ventral parts of the pons. The patient suffers from hemiplegia, with some paresis on the opposite side to the hemi-

FIG. 213.—Lesions of the midbrain and resulting symptoms. 2. Hemianopsia; 3. disturbances of pupillary light-reflex; 4. ipso-lateral paralysis of eye-muscles, contra-lateral hemianæsthesia, forced movements, tremors; 5, as in 4 plus crossed hemiplegia; 6, crossed motor and sensory disturbance with slight oculo-motor symptoms on same side; 7, one-sided or double-sided oculo-motor paralysis, complete dilatation of pupils, amblyopia; 1.8, hemianæsthesia, central pain, visual hallucinations.

plegia. There is great exaggeration of reflexes and twitching spasms and coarse, irregular, jerky movements of the limbs, but no convulsion. There is usually anæsthesia and ataxia on the side of the lesion, and the anæsthesia is dissociated. There is often forced weeping and laughter and dysarthria may occur. Forced movements are not common. When the lesion is high up and involves some of the transverse branches of the basilar, we get oculo-motor palsies and spasmodic disorders on the side of the lesion and paralysis and anæsthesias on the opposite side. When lower down, we get fifth and seventh nerve palsies on one side and hemiplegia on the other.[1] In basilar artery lesion, if the injury is large and severe, the patient develops disorders of deglutition and speech and

[1] *Foville's Paralysis.*—A "crossed hemiplegia" with paralysis of the sixth and seventh nerves on the side of the lesion and paralysis of the extremities on the opposite side.

extension of the process and dies from paralysis of the bulbar centres. No great disturbances of temperature occur, nor are there marked pupillary symptoms. In the smaller lesions due to involvement of the transverse branches recovery may occur, leaving the patient with a crossed paralysis.

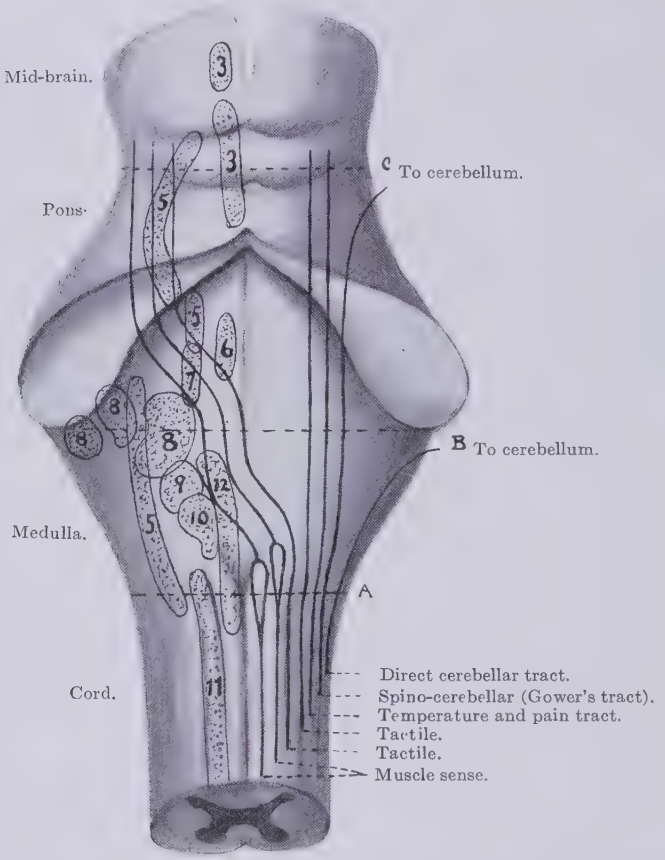


FIG. 214.—Showing how lesions in medulla and pons may cause focal symptoms by cutting off tracts and destroying cranial nerve nuclei. The motor tracts lie deep below the sensory and begin to cross in the lower medulla and cord. They are not indicated here.

When the lesion is such as to cause paralysis of the facial nerve on the side of the lesion and hemiplegia on the opposite side, we have what is called the *Millard-Gübler syndrome.*

Softening of the Medulla.—Softening is rare, but much more frequent than hemorrhage. It occurs at the early apoplectic age—thirty to fifty, oftener in males, and is due to lues and the arterial changes associated

with alcoholism and nephritis. It has been observed as a result of working in compressed air.

A small lesion may cause tetraplegia; or it may be low down involving the leg fibres of the pyramidal tract after they have crossed and the arm fibres before they have crossed. This causes paralysis of the arm on the contralateral side, of the leg on the homo-lateral side (hemiplegia cruciata).[1]

A rather special syndrome is caused by thrombosis of the inferior cerebellar artery. The symptoms may come on slowly almost like a tumor. Finally a seizure occurs with symptoms which vary somewhat, but are about as in the following table:

Homolateral side	Contralateral side
Paralysis	Hemiplegia.
of soft palate	Cutaneous anæsthesia for pain and temperature.
of vocal cord	
of Trigeminus	
of facial	
of sympathetic	
Cerebellar ataxia +++	Ataxia?

The cause of the symptoms is shown in Fig. 214. The tracts for muscular and tactile sensations have not altogether crossed in the medulla, hence we may get some evidence of ataxia on both sides. But the cerebellar tracts and peduncle injured have not crossed.

CHRONIC PROGRESSIVE SOFTENING OF THE BRAIN

Sometimes thrombotic processes affect especially and successively the smaller central arteries of the circle of Willis and lead to a gradual softening. With this process there develops a gradual hemiplegia coming on perhaps in stages during the course of six months or a year. With the hemiplegia there may be headaches, vertigo, paræsthesia and anæsthesia, dysarthria with some mental deterioration. The progressive character of the disease suggests a brain tumor or abscess. In very severe cases there may be optic neuritis and atrophy. Usually, after reaching a certain stage, the disease is arrested and the patient remains about the same for some years.

[1] *Hughlings-Jackson's syndrome* is a condition produced by medullary lesions in which there is hemi-atrophy of the tongue and paralysis of the trapezius, sterno-mastoid, vocal cord and soft palate, all on the same side.

Senile hemiplegia.— Marie has described a condition known as *lacunar degeneration.* It is characterized by the presence of cavities of various sizes ($\frac{1}{10}$ to $\frac{3}{4}$ inch) and irregular contour distributed through the brain. Through these cavities runs a sclerotic but permeable blood-vessel. The softening is not thrombotic but due to the disease of the vessel wall and interference with the nutrition by the ordinary peri-vascular exchanges. The presence of lacunar degeneration leads to attacks of hemiplegia which are usually mild and sometimes temporary. They may not be accompanied with loss of consciousness or abolition of the skin reflexes. Lacunar degeneration thus causes a hemiplegia of the aged.

Lacunar degeneration, associated with arterial sclerosis is one of the factors in chronic senile paraplegia, characterized by short-step gait (*dèmanche à petit pas*).

THROMBOSIS OF THE CEREBRAL SINUSES

Sinus thrombosis may be non-inflammatory or inflammatory. Non-inflammatory thrombosis is due to a weak heart and usually occurs in children under the age of one year, in very old people, or at the end of exhausting diseases.

Inflammatory sinus thrombosis is due to some neighboring bone disease, especially otitic disease. However, any disease of the bones of the skull and especially of the orbital and nasal cavities, may give rise to the thrombosis as nearly all the sinuses receive a small venous supply from the exterior of the skull.

Sinus thrombosis from exhausted conditions and weak heart affects usually the longitudinal sinus. Otitic thrombosis affects the lateral (transverse) superior and inferior petrosal sinuses. Orbital thrombosis affects especially the cavernous sinus.

The general symptoms of sinus thrombosis vary greatly according to the part affected, the extent of the obstruction, the primary cause, and the complications. There are, therefore, symptoms of cardiac weakness and marasmus, or of otitic, orbital, nasal or other cranial bone disease, with corresponding local and general symptoms of infection.

The cerebral symptoms vary also but resemble those of an acute meningitis, or of a focal hemorrhage or softening. There occur headache, vomiting, stiff neck, delirium, coma, convulsions, hemiplegia, aphasia. There is rarely temperature from the thrombosis alone.

Some of the special sinus symptoms are as follows:

In cavernous sinus thrombosis, there are dilated facial veins, protrusion of the eyeballs with orbital œdema, due to swelling of the retro-bulbar veins, sometimes choked disc, supra-orbital pain and involvement of the motor nerves of the eye (Fig. 215).

In thrombosis of the lateral (transverse) sinus, there is swelling and œdema over and about the mastoid, with tenderness in this region and along the jugular vein, in which a clot may be felt. There is pain locally and on lateral movement of the head, and on swallowing. If the thrombosis is not septic or complete and solid the subjective symptoms may be slight.

If it is a septic thrombosis there are symptoms of general infection, and of cerebral involvement. These latter most often resemble those of meningitis, though lumbar puncture will not show lymphocytosis.

Fig. 215.—Exophthalmus, thrombosis of cavernous sinus. (*Weldebrandt and Sanger.*)

In thrombosis of the longitudinal sinus there is swelling especially of the frontal and parietal veins, causing the *caput Medusæ;* sometimes nose-bleed, and in infants distended fontanelles.

The prognosis in primary thrombosis is not good, but cases may recover. In infective thrombosis, the prognosis is very serious unless there can be surgical interference.

The treatment of sinus thrombosis is symptomatic and surgical. Its details are largely in the hands of otologists, rhinologists and general surgeons.

CEREBRAL PALSIES OF CHILDREN—INFANTILE HEMIPLEGIA AND DIPLEGIA

The brain palsies of early life show themselves in the form of (1) hemiplegias; (2) diplegias or double hemiplegias, in which both sides of the body are involved; and (3) paraplegias, in which the lower limbs

are chiefly or entirely involved. In these palsies, as in the same troubles of adult life, the loss of motor power is always accompanied by a rigidity and by some contractures and exaggeration of reflexes, in this respect distinguishing these paralyses from those of spinal origin. The seat of lesion in these cases is in the hemispheres of the brain, and it is the upper motor neurons which are involved; that is to say, that part of the direct motor tract which extends from the brain cortex down to the spinal cord as far as the anterior horns. The brain palsies of children are therefore disorders of the cortico-spinal neurons, while the spinal palsies of children are disorders of the lower neurons.

I have reserved the term Little's disease for those cases of cerebral diplegia due to agenesis of the pyramidal tracts.

Etiology.—The disease occurs rather oftener in males than in females, though the difference is slight. The vast majority occur in the first three years of life; about one-third of them are congenital. Injuries to the mother during the time of pregnancy, possibly diseases and emotional disturbances at this time, are factors in producing the congenital cases. Those cases that occur at the time of birth are due to tedious labor, the use of forceps, and other injuries at the time of parturition. After birth, the causes are those which lead to the production of intracranial hemorrhages, embolism, and thrombosis; these being injuries and the infectious fevers. Of the latter, pneumonia, encephalitis, whooping-cough, measles, and scarlet fever are the most prominent. Syphilis is a cause in 15 to 20 per cent.; cerebrospinal meningitis and epileptic convulsions are also occasional causes.

Symptomatology.—The disorder in about one-fourth of the cases begins with a convulsion, which may be unilateral, but is usually general in character, and may last for several hours. At the same time a febrile process develops, and this continues for several days. When these acute symptoms have subsided, or before this, it is noticed that the child is paralyzed upon one side, the paralysis involving the arm, leg, and face, as in adult hemiplegia, or perhaps involving both sides. This paralysis undergoes gradual improvement, the face recovering earliest and most, the leg next, and the arm least. As the child develops it is found that the paralyzed side fails to grow as fast as the other, and there may be from one-half to one inch or two inches of shortening in the arm or leg. The circumference of the limbs is less, the surface somewhat colder, and some vasomotor disturbance may be present. With the progress of the case a rigidity of the affected limbs develops; the heel becomes drawn up, so that there is talipes equino-varus or equino-valgus. The flexors of the forearm and of the wrist and fingers contract, as do also the adductors of the thighs. In general it will be found that there is a contraction of the flexors and adductors of the affected limbs. With this rigidity and the

contractures there are exaggeration of reflexes and clonus in most cases. In the disordered limbs peculiar mobile spasms develop. These consist of athetoid, choreic, and ataxic movements, also sometimes tremors and associated movements. The choreic and athetoid movements are the most common.

Along with the appearance of these symptoms it is noticed that there are disturbances in the mental condition of the child. It is usually backward in development, this backwardness ranging from simply feeble-mindedness to complete idiocy. Taking all cases, there is about an equal division between feeble-mindedness, imbecility, and idiocy (Sachs). Perhaps a little over one-fourth of the subjects have a fair intelligence. There is usually slowness in learning to talk, and in a small proportion of cases there is a decided aphasia. Such condition is rather more frequent with right hemiplegia than with left hemiplegia, though the rule is not an absolute one. In connection with the mental defect there may develop many of the peculiar moral traits associated with idiocy and low degrees of intelligence. Epilepsy very frequently complicates the disease; nearly one-half of the subjects suffer from this trouble. This epilepsy is in most cases general in character; in a few cases it takes the Jacksonian type, in a small number *petit mal* alone is noted. Examination of this class of sufferers reveals, aside from the paralysis described, various evidences of defective development. These are known as stigmata of degeneration; though they cannot be classed strictly among such, since they are acquired stigmata in most cases, rather than marks which are the result of primary deficiency in development. These stigmata consist of a microcephalic or a macrocephalic skull, cranial and facial asymmetry, prognathism, imperfectly developed teeth, and a high palatal arch. It has been found that, as a rule, in cases of cerebral hemiplegias of childhood the patient eventually has a slight flattening of the skull on the side of the lesion (Fisher and Peterson). Finally, in a few cases there may be found defects in the special senses, such as imperfect hearing, deafness, deaf-mutism, and defects in vision, such as hemianopsia, and perhaps imperfections in smell and taste. Anæsthesia is never observed.

The symptoms in the cerebral palsies of children, having passed the acute stage and having become somewhat ameliorated, enter into a chronic stage. This chronic stage begins within a few months after birth or after the onset of the disease. No great change occurs in the paralyses as the child grows older until he reaches the time of puberty, though there is a slight improvement in most cases. After the time of puberty, if the mental condition of the child is good, the physical symptoms are apt to improve considerably.

Morbid Anatomy.—The primary changes that lead to the cerebral palsies of children are: 1st, simple agenesis or lack of brain development,

producing localized atrophy of the cerebrum and the condition known as *porencephalus*, a condition in which, owing to a congenital defect in nutrition, a cavity or depression exists in the cerebral hemispheres, this cavity reaching generally into the lateral ventricle. True porencephalus is found in about one-fourth of the cases, though no definite statistics can be given, owing to the different interpretations given to this term; 2d, hemorrhage, which is probably the most frequent of the single causes; 3d, embolism; 4th, thrombosis; 5th, meningo-encephalitis and polio-encephalitis; 6th, a diffuse cortical sclerosis. Many other terms are used to describe the pathological conditions found at the basis of the

FIG. 216.—Atrophied brain with sclerosis and a cyst, from a case of infantile cerebral hemiplegia.

brain palsies of children, but the principal causes of all are undoubtedly, as has been described, hemorrhages, encephalitis and softenings, and a defective development or agenesis. It is probable that in the hemiplegies the original lesion is generally a central hemorrhage, less often a meningeal hemorrhage. After this, probably the most frequent condition is a porencephalus from some intra-uterine accident, which may have been defective nutrition causing anæmia and softening, or hemorrhage, or thrombosis. Acute encephalitis or inflammation of the brain of the kind similar to poliomyelitis is a frequent cause. In double hemiplegias or diplegias of children the cause is in the vast majority of cases a hemorrhage due to some injury or disturbance at the time of labor.

In other cases of diplegia the lesion is a double porencephalus, which may be either the result of an intra-uterine hemorrhage or simply a defective development. In the paraplegias the lesion is probably very much the same as in the diplegias, that is to say, either a meningeal hemorrhage or a brain agenesis. Occasionally a diffuse sclerosis has been found in these cases. Not infrequently, as the result of hemorrhages, there develop cysts which fill up the atrophied areas of the brain (Fig. 216). It is difficult to present accurately and definitely the relations between the pathological change and the clinical result, but it may be shown with some degree of correctness in the following table.

Original Lesions	Later Pathological Conditions	Clinical Result
Hemorrhage.	Atrophy.	Hemiplegia.
Embolism.	Lobar and diffuse sclerosis.	Diplegia.
Thrombosis.	Cysts.	Paraplegia.
Agenesis.	Porencephaly.	Sensory defects.
Encephalitis.	Microcephaly.	Mental defects.
		Epilepsy, etc.

Diplegia or Birth Palsies.—That form of the brain palsies of childhood characterized by double hemiplegias or diplegias has certain special characters which lead to its being often classed apart. These diplegias in almost all cases are congenital and are due either to injuries at the time of birth or to some disorders of intra-uterine life. There may be convulsions or a prolonged state of asphyxia at the time of birth. After recovery from this no special trouble is noticed with the child by the mother for some weeks or months, when it will be found that it does not use its arms or legs. Other convulsions develop, and eventually the features of a double hemiplegia with mental impairment and epilepsy are observed. In these cases the mental defect is much more decided than in the hemiplegias; indeed, few of these cases ever show any good amount of intelligence. Epilepsy is extremely common. The anatomical lesion in the cases is, as already stated, either a meningeal hemorrhage which has pressed upon and injured the cortical motor areas in each hemisphere, or it is a congenital porencephalic defect. (See Little's Disease.)

Diagnosis.—The clinical diagnosis of cerebral palsies is to be made from the spinal palsies. The latter are distinguished by the fact that in the infantile paralyses of spinal origin there is no rigidity or exaggeration of reflexes, and there are electrical degenerative reactions of the muscles and decided wasting of the limbs with shortening. The mode of onset in cerebral palsies and their distribution in the form of hemiplegias in which the face is involved also indicate the seat of the lesion. The pathological diagnosis is by no means an easy one. Cerebral palsies occurring at the time of birth and accompanied at the time by general convulsions or asphyxia may be considered to be due to meningeal hemorrhage, especially if the delivery of the child has been brought about by the use of

forceps or if the labor has been long and tedious. Diplegias and paraplegias which are congenital are probably due to true porencephalus, provided there was no difficulty at the time of labor and there were no convulsions or other serious phenomena after it. Cerebral palsies occurring after birth in the first, second, or third year of life are apt to be due to hemorrhage and encephalitis, less often to embolism or thrombosis. Hemiplegias developing after infectious fevers are likely to be due to hemorrhage. In diagnosticating the pathological lesion in such cases it must always be remembered that hemorrhage is much more frequent than embolism, and that thrombosis occurs only in children with very weak hearts and in marasmus.

Course and Prognosis.—In all types of the disease the course is chronic and perfect cure is hardly possible, although in the slighter forms of hemiplegia nearly all traces of the paralysis may be absent. In the hemiplegic form the patient often reaches adult life, and if his intelligence is not defective and he has no epilepsy the motor trouble improves a great deal and he may live a long and useful life. If epilepsy and mental defect are present, there ensues eventually a further mental deterioration, and such subjects rarely live much beyond the period of adolescence, or if they do they pass into the asylums for the idiotic and epileptic. The diplegic and paraplegic cases have a much worse prognosis both as to duration of life and as to improvement in symptoms, except occasionally the type described above as due to agenesis of the pyramidal tracts. The degree of intelligence and the absence of epilepsy are the two factors which measure the seriousness of these cases, as they do those of the hemiplegias. As regards the significance of individual symptoms, the post-hemiplegic movements have a bad import; the presence of a microcephalic head or of decided marks of degeneration is unfavorable.

Treatment.—The treatment, so far as the paralysis is concerned, is largely mechanical. The patient is benefited by occasional courses of electrical treatment which stimulate somewhat the nutrition and functions of the muscles. Massage and stretching of the contractured tendons and limbs also are helpful in my experience. The orthopædic surgeon is able to render valuable assistance by occasional overstretching the contractured limbs and placing them in splints. Tenotomy may also be resorted to with advantage, as I have had occasion to see. The child should be encouraged above all, however, to use the limb as much as possible. He should be taught gymnastic exercises; running, walking, and bicycle riding are all measures which give great help. When the child's intelligence is good and there is little or no epilepsy, a great deal can be expected in the way of improvement as he grows older.

So far as the epilepsy is concerned, it should be treated on the same principles as idiopathic epilepsy, except that great care should be had in

the use of the bromides; a thorough test must be made in order to determine how much of this drug will suppress the fits, and then its use must be graduated in the future in accordance with the knowledge thus obtained. The mental defects of the child can be helped only by proper training of the body and careful education of the mind. The question of operative interference in these cases has excited much attention. *A priori* it would not seem as though surgical interference could do good in relieving conditions in which there is destroyed or atrophic tissue. Still the subject must be dealt with empirically, and there have been some results which show that apparently a relief is obtained in a few cases by trephining the skull and if he then finds any evidences of compression from the presence of a cyst, this may be very cautiously opened. Resection of the posterior spinal roots has been tried with some successes reported.

CHAPTER XIX

TUMORS OF THE BRAIN

All forms of new growths are found in the brain, but the infectious granulomata, tubercle and gumma, and the sarcomatous type of tumors are the most common. Cushing found that 64 per cent. of brain tumors were gliomata and gliomatous. Elsberg's statistics show about the same proportion.[1]

[1] In order to present a working view of brain tumors from a surgical standpoint, Dr. Charles A. Elsberg has kindly furnished me an analysis of 100 cases subjected to radical, exploratory or palliative operation. The figures naturally are not the same as those derived from a series of post-mortem studies like those of Schlessinger. but they represent more accurately the kind of cases that come to the neurologist, I present only the salient features of his analysis.

Location	
Cerebral	44
Cerebellar or pontine	28
Unlocalized	20
Cortical or extra-cortical	14
Deeply subcortical	58
Not localized	28
Frontal, central and parietal	21
Occipital and temporal	5
Thalamic, 3d ventricle and mid-brain	10
Hypophysis and peduncular region	8
Cerebellum	20
Cerebello-pontine angle	7
Pontine	1
Nature of Growth	
Glioma	33
Gliomatous cyst	3
Gliosarcoma	1
Sarcoma	3
Endothelioma	5
Adenoma	5
Teratoma	1
Cholesteatoma	1
Neurofibroma	5
Simple cyst	2
Nature unknown (no examination)	13
Nature unknown (unlocalized)	28
	100
Operative Result	
Tumor was removable and removed in	13
Tumor was partly removed in	5
Cyst (non-gliomatous) was evacuated in	2
Tumor could not be removed on account of nature or location, or the tumor was not localized in	80
	100

Etiology.—Brain tumors affect males oftener than females, the ratio being about as two to one (644 : 320). Sarcomata alone seem to affect females about as often as males. Brain tumors occur with about equal frequency throughout all ages of life up to about fifty; one-third occur under the age of twenty (Gowers). During childhood tumors are about equally distributed throughout all ages (Starr). One-half of all the tumors of childhood are tuberculous; after this come gliomata, endotheliomata and sarcomata. The gumma, glioma and sarcoma begin to be more frequent after the age of twenty. Sarcoma and especially cancer occur in the middle and later ages of life; but brain tumors of any kind are extremely rare after the age of sixty.

To sum up in tabular form, the relative frequency of the different kinds of tumors with regard to age is shown in the following:

Childhood,	tubercle, parasites.
Early life,	gumma, glioma, parasites.
Early and middle life,	sarcoma, endothelioma, glioma, and gumma.
Middle and late life,	sarcoma, endothelioma, gumma, cancer.

Heredity has a slight influence in predisposing to brain tumors. Blows on the head and other forms of injury to the cranium are exciting causes in a small proportion of cases. Practically all cancers and many sarcomata of the brain are metastatic in origin.

Seat.—About one-fourth of all tumors are in the cerebellum, or cerebello-pontine angle, and about one-fourth are in the frontal lobe. About one-twelfth of all tumors are in the basal ganglia and lateral ventricles, and a rather smaller number in the mid-brain and medulla. Tumors more often lie in the cortex and parts just below, than in the centrum ovale.

In children the locations are a little different. Here nearly one-half of the tumors are in the cerebellum and pons. They are very much less frequent in the cortex cerebri but more common in the centrum ovale, basal ganglia and mid-brain. As the mid-brain as well as all the contents of the posterior fossa are nourished by the vertebral arteries and their branches, it follows that the majority of tumors in children are in this vascular area.

Symptoms.—The symptoms of brain tumors vary extremely in accordance with the location, the kind of tumor, the rapidity of growth, and the age of the patient. The general course of a case of brain tumor in an adult is somewhat as follows: The patient first notices a headache which is intense and persistent, and which has exacerbations of frightful severity. With the headache or between the attacks vomiting occurs, which is often not accompanied by any nausea. Sensations of vertigo, annoying paræsthesias, and convulsive movements affecting one or more

This indicates that about 20 per cent. of brain tumors brought to a special hospital or neurological surgeon are operable.

extremities develop, and there may even be general convulsions. Papillœdema and optic neuritis may then appear and the patient finds that his eyesight is weak and progressively deteriorating. The mind becomes more or less disturbed, the mental processes are dull and slow, a feeling of hebetude and incapacity to attempt any mental exertion are present. As the disease progresses the intense pains and vomiting produce weakness and emaciation. Paralyses of various kinds develop and blindness ensues. Convulsions of a local or general character become more frequent, and finally the patient becomes bed-ridden and helpless.

The course of the disease is not a steady one, there being often slight remissions, or there may be periods when progress seems to be arrested. After a period of time varying from one to four or five years death occurs from exhaustion or some intercurrent malady.

The symptoms thus very briefly outlined are divided into general and focal. The *general symptoms* are:

Headache.
Vertigo.
Vomiting.
Papillœdema.
Mental defects.

Besides these there may be general convulsions and speech disturbances.

Headache occurs in from one-half to two-thirds of the cases; it is very severe and the pains are of a boring or lancinating character; they are so horrible that they often lead the patient to think of suicide. The pains are sometimes periodical, occurring every night or every other day, and suggest by their periodicity a malarial character. They are located sometimes in the brow or in the occiput, while sometimes they are diffused all over the head; they are rather more frequent than otherwise in the neighborhood of the tumor. They are more frequent with cerebellar tumors than with those located anywhere else. They are less frequent with tumors situated in the peduncles and at the base of the brain. The pains are due to the increased intra-cranial pressure and to irritation and stretching of the dura mater by the encroachments of the new growth. Headache occurs in about the same proportion in children and adults, and it does not seem to bear much relation to the kind of tumor, although the pains are generally less with the gliomata, and they are more frequent with rapidly growing tumors whatever their character. With the pains there is often a local tenderness of the scalp and cranium which may be elicited by percussion, and in most cases there is greater tenderness in that part of the cranium lying over the tumor.

Vomiting is a symptom which is almost as frequent as headache. The vomiting is often of a projectile character and not accompanied by

much nausea. Vomiting occurs, as does headache, more frequently with cerebellar tumors. It is associated with rapidly growing tumors, such as syphilitic or tuberculous neoplasms.

Vertigo is a general symptom which occurs in from one-third to one-half of the cases. The vertigo may be slight, such as is often felt from ordinary causes. Occasionally it is very severe and accompanied by forced movements. The severer forms and those associated with forced movements occur with tumors of the cerebellum and the parts closely connected with it.

Papillœdema or choked disc is one of the most frequent and important of all the general symptoms of brain tumor; it occurs at some period of the disease in at least four-fifths of the cases, more frequently in cerebellar tumors and in those of the mid-brain and great basal ganglia. It is rare in tumors of the medulla. It is less frequent and marked in the slow-growing tumors and in infiltrating gliomata of the hemispheres. The condition may run a somewhat rapid course and then improve a great deal or even for a time disappear; but ordinarily the course is progressive and it ends eventually in neuritis and an atrophy of the optic nerve. Hence the examination of the eyes in brain tumors should be made a number of times in order to note the progress of the trouble. Primary atrophy of the optic nerve does not occur in brain tumors, except to a certain degree in some frontal tumors.

The choked disc is caused mainly by the pressure of the fluid in the subarachnoid space, which space communicates with the connective-tissue sheath of the optic nerve. This presses the nerve upon the lamina cribrosa, the central vein of the retina is strangulated and œdema and swelling follow. (Parker states that the choked disc is due to a disproportion between the intra-ocular tension and the intra-cranial pressure, by reason of which the disc is pushed into the eye.) Generally there is a greater degree of papillœdema on the side of the tumor.

Slight mental defects are almost always present in tumors of the brain. These defects consist in a slowness of the mental processes, a condition of hebetude, a tendency to attacks of somnolence, and sometimes a peculiar childishness, silliness and tendency to foolish joking. The memory is also usually somewhat weakened and the power of attention lessened. Such psychical defects are more frequent with tumors of the frontal lobes and more frequent also with large tumors.

General convulsions occur in about one-fourth of the cases and more frequently when the tumors are situated in the cerebral hemispheres and cortex. There may be also apoplectiform attacks, from which the patient recovers in the course of a few days or weeks. More rarely there is a genuine apoplexy from the bursting of a blood-vessel in the neighborhood of the tumor.

The local symptomatology of cerebral tumors cannot follow always strictly the anatomical subdivisions of the brain. There are symptoms distinguishing tumors which lie above and those which lie below the tentorium because the pressure conditions are not alike.

Fig. 217.—Showing relationships of tumor in the posterior fossæ of the skull.

In *subtentorial tumors* pressure soon blocks the aqueduct of Sylvius and causes a hydrocephalus and pressure symptoms. Tumors of the hemispheres cause the change more slowly, consequently optic neuritis and the general brain symptoms of tumor may be less marked.

1. **Tumors of the Prefrontal Area.**—Tumors in this area may show no localizing symptoms. Nevertheless in a good proportion of cases something characteristic may be observed. Sometimes there is mental disturbance, which is mainly of the nature of mental deterioration, perhaps it simulates a dementia paralytica, or there may be only a certain degree of foolishness and silly jocosity (Witzelkeit). The pressure of a frontal tumor upon the olfactory nerves may cause disturbances in the sense of smell. Pressure on the optic nerve may cause disturbances of the visual field and papillœdema. But the pressure on the optic nerve being local and direct may produce at the same time an early atrophy of the central fibres shown by the presence of a *central scotoma* (Kennedy). Rarely, the tumor involves the orbit causing paralysis of the ocular muscles and protrusion of the globe of the eye. If the tumor grows backward there is gradual invasion of motor centres with irritation, showing itself by local convulsions, and later by paralyses.

Occasionally in frontal tumors there is tremor of the hands, and a kind of ataxia of gait and station (frontal ataxia).

2. **Tumors of the Precentral Convolutions.**—It is in this area that we are often able to make the closest and most accurate diagnosis of the localization of new growths, owing to the involvement of the different motor centres. These centres are at first irritated, with the result of producing local spasms or Jacksonian epilepsy. Such spasms are often preceded by sensory symptoms or auræ. As the tumor grows, the area of involvement becomes larger, spasms become more diffused, and general convulsions may finally appear, with hemiplegia. Besides the symptoms mentioned, there may also be motor aphasia and agraphia. If the tumor is subcortical the symptoms are more of the paralytic and spastic type and less of a convulsive nature than when the cortex is implicated.

3. **Tumors of the parietal lobe** show disturbances of sensation and of perception tactile, muscular and visual. Tumors in the anterior portion (post-central convolution) cause disturbances of cutaneous sensation; those in the superior lobule cause astereognosis; those in the inferior lobule, anteriorly loss of deep muscular sense with ataxia; those further back cause forms of visual imperception such as apraxia and if on the left side, alexia.

If the tumor lies deep there may be hemianopsia. If the tumor is in the post-central we get with disturbance of cutaneous sensation, usually some motor phenomena due to irritation of the precentral.

4. **Occipital Lobes.**—Tumors of this region, if situated in the cuneus and calcarine fissure, produce homonymous hemianopsia partial or complete, the macular region escaping. If the tumor involves the other parts of the left occipital lobe and the cuneus is not seriously involved, there

may be a condition known as soul blindness, sensory apraxia or imperception consisting in an incapacity to understand the nature of the things which one sees. If the tumor extends up chiefly toward the angular gyrus, on the left side, there may be word blindness, along with some hemianopsia. If the tumor extends farther forward and deeply into the parietal lobe, there will be hemianæsthesia, hemiataxia, and perhaps a little hemiplegia owing to involvement of the fibres of the internal capsule.

Hallucinatory symptoms may occur in occipital lobe tumors.

5. **Temporal Lobes**—The temporal or temporo-sphenoidal area on the right side is very nearly a latent one. On the left side, tumors involving the posterior part of the first and upper posterior part of the second temporal convolution produce sensory aphasia.

Tumors of the anterior and inner side of the lobes involving the uncinate and hippocampal gyri may produce dreamy states and disturbances of smell and taste (uncinate fits). Tumors lying further back may cause also third, sixth and seventh nerve trouble if they lie in the external portion, but if posterior and internal may cause nystagmus and cerebellar symptoms. As the tumor gets deeper it may cause hemianæsthesia and hemiplegia, the symptoms of paralysis being more marked and earlier in the face and arm (Kennedy).

6. **Tumors of the Corpus Callosum.**—Tumors situated in this area are very rare. Their symptoms are somewhat characteristic and correspond somewhat with tumors situated in the ventricles of the brain; in other words, tumors which, beginning in the central parts of the brain, gradually extend outward toward the periphery. The symptoms credited to tumors of the corpus callosum are mental dullness, stupidity, dyspraxia and drowsiness, the patient often sitting for hours mute, refusing to speak, or lying in a half-somnolent condition. There are no paralyses of the oculo-motor or other cranial nerves and often no optic neuritis, or any severe headache and vomiting. There is no anæsthesia. The symptoms progress; locomotion is early impaired and later the patient becomes unequally diplegic. The disease gradually progresses and the patient dies in coma.

7. **Tumors of the Great Basal Ganglia and the Capsule (the opticostriate region).**—The general symptoms of tumors of this region resemble in many respects those of tumors of the corpus callosum. The hebetude, however, may be less marked. There is usually a progressive hemiplegia which may be accompanied by anæsthesia and sometimes by choreic movements, if the tumor involves the optic thalamus and adjacent part of the capsule.

Tumors of the corpus striatum cannot be diagnosticated by symptoms due to the irritation or destruction of this ganglion. The location can

be only approximately estimated by the presence of symptoms due to lesion of the adjacent parts, viz., the internal capsule and the adjacent motor cortex. When on the left side, symptoms of aphasia of the motor type may appear. As the lesion extends there may be hemiplegia, and later, perhaps, irritative symptoms from involvement of the capsule and motor cortex.

Tumors of the Optic Thalamus.—The same may be said of tumors here as of those of the corpus striatum. Lesions of the thalamus itself

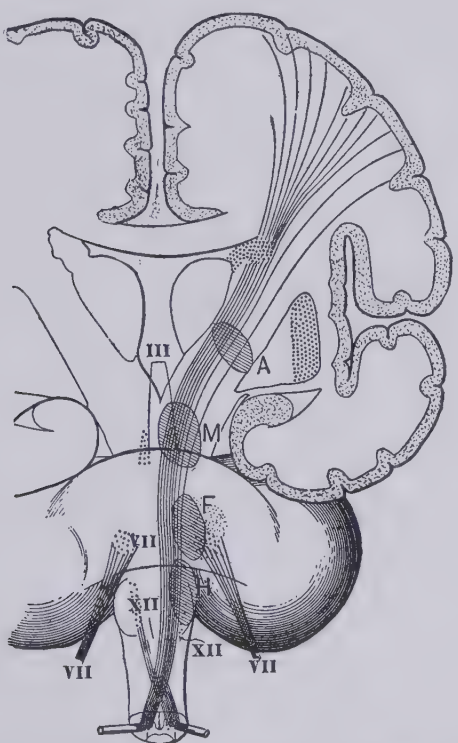


FIG. 218.—Showing the mechanism of crossed paralysis. Lesion at *M* causes paralysis of third nerve, lesion at *F* paralysis of fifth nerve with hemiplegia of opposite side.

give no specific symptoms unless it is that of pain referred to the extremities of the opposite side. But by extension we get hemianæsthesia of various types and involvement of the ocular muscles and reflexes. There is later even homonymous hemianopsia; peculiar forced movements and intention tremor may occur. The opinion that in lesions of the thalamus there is a paralysis of emotional expression has been sustained by Mills. The thalamic syndrome which is observed in acute lesions of

this ganglion does not appear very characteristically in chronic progressive lesions.

Tumors of the Pineal Gland, the Pituitary Body and Optic Chiasm.—The thalamus and adjacent region ('tween-brain or diencephalon) is a part of the brain which in man is not sharply limited. It may be divided into an upper or dorsal part, including the thalamus, geniculate bodies and pineal gland, and a lower or ventral part, hypothalamus, which includes the corpora mammillaria, the pituitary body and infundibulum and the optic chiasm. Tumors of the pineal gland are generally teratomata or psammomata. They may cause disturbance of metabolism leading to adiposity and precocious mental, physical and sexual development. Secondarily, they cause neighborhood symptoms such as disturbance in ocular movements, optic neuritis, ataxia, and hydrocephalus from blocking the aqueduct of Sylvius.

Tumors of the pituitary body may be associated with giantism if they occur before adolescence and ossification of the epiphyses, and with acromegaly if they occur after this period. As they grow they press on the chiasm and cause optic neuritis and atrophy and a characteristic bitemporal hemianopsia. The pituitary disease may lead to other disturbances of nutrition such as adiposity and increased sugar tolerance, andchanges in secondary sexual characters forming the Fröhlich syndrome. (See Acromegaly.)

Tumors of the Mid-brain.—The mid-brain includes the cerebral peduncles, the corpora quadrigemina and the aqueduct of Sylvius. In the base of the peduncles run the cerebrospinal tracts, above which is the locus niger and above this the tegmentum with sensory tracts, the anterior cerebellar peduncles, the red nuclei and the nuclei and motor nerves of the eye (Fig. 213). The anterior tubercles of the corpora quadrigemina and geniculate bodies have to do with the reflexes and coordination of the eye movements, the posterior with the functions of the eighth nerve. Tumors here are sometimes tumors of the peduncles, and sometimes of the corpora quadrigemina, according as they are superficial or deeply situated. The general character of the symptoms is an association of hemiplegia with disturbances of the motility and reflexes of the eye, often ataxic symptoms and sometimes forced movements and disturbances of hearing. We would expect soon to get symptoms of ocular palsies and of brain compression from closure of the aqueduct of Sylvius.

As many as six different groups of symptoms or *syndromes* have been described due to lesions here. Some of these are given under the head of acute softenings and hemorrhages. (See p. 443.)

When a mid-brain tumor lies in a cerebral peduncle it produces "alternate hemiplegia." Thus if the tumor is in the anterior portion and

situated high up, there is a palsy of the third nerve on one side and a hemiplegia of the opposite side (syndrome of Weber). If it is in the posterior portion and a little lower it will cause third nerve paralysis on one side and tremor or choreic movements on the other (syndrome of Benedict).

Tumors of the corpora quadrigemina cause pupillary inequalities and paralyses, oculo-motor paralyses, vertical and lateral nystagmus. If deep enough to irritate the cerebellar peduncle, forced movements, asynergy, and disturbance of equilibrium.

Tumors of the pons cause paralyses of cranial nerves on one side and hemiplegia on the other. If the tumor lies near the fifth nerve, there may be a palsy of this nerve on one side and hemiplegia on the other side (Fig. 218). If the tumor is larger it may produce paralysis of the fifth,

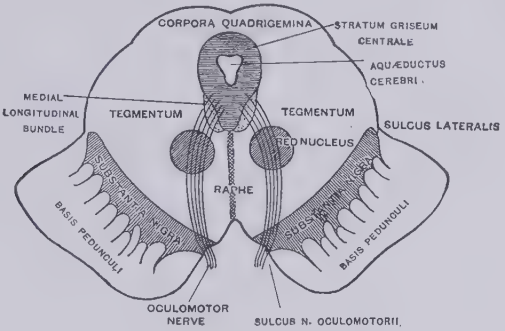


Fig. 219.—Section through mid-brain.

sixth and seventh nerves on one side and hemiplegia on the other (syndrome of Gubler). If situated somewhat superficially and on the lateral edge of the pons involving the peduncles, there will be forced movements of the body, either toward or from the seat of the lesion. An important sign of intra-pontile tumor (Spiller) is paralysis of lateral associated movements of the eyes. If the tumor is unilateral the eyes cannot be turned to the side of the lesion.

Tumors of the medulla are rare and generally tubercular or syphilitic. The medulla is the seat of origin of the IX, X, XI in part and XII nerves. Through it run motor and sensory and cerebellar tracts. It is the centre for the automatic regulation of respiratory, cardiac and vasomotor functions. The olivary bodies place it in close relation also with the cerebellum. Tumors here cause, therefore, paralyses of the tongue, disturbances of deglutition, speech and finally of respiration and cardiac action. Hemiplegia, hemianæsthesia and ataxic symptoms may develop.

Tumors of the cerebellum involve often a study of the condition of the vestibular nerve, of the spinal apparatus of equilibrium, and of

the cerebro-cerebellar connections. It involves the employment of the diagnostic tests previously given. The accompanying diagram of the cerebellar connections, modified from Thomas and made more complete by Dr. W. F. Schaffler, will be found useful.

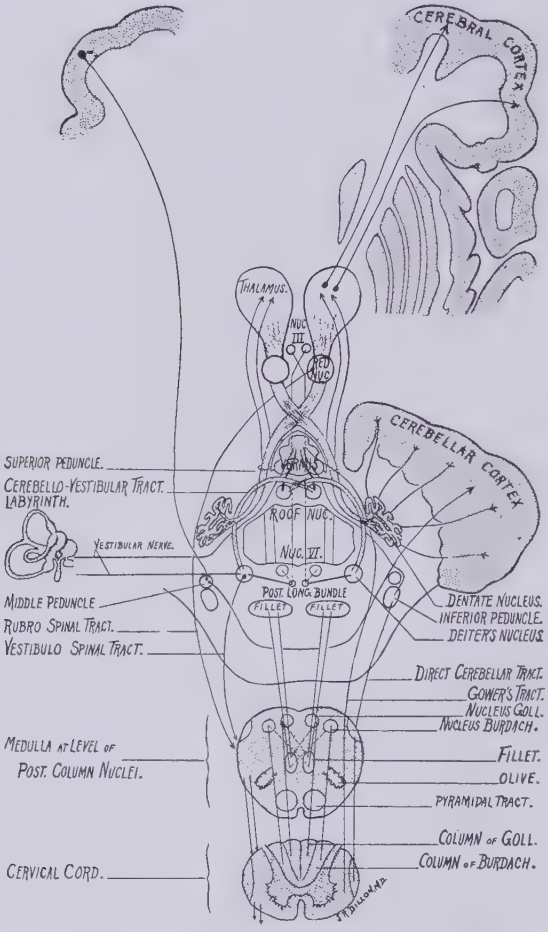


FIG. 220.—Connections of the cerebellum with cerebral cortex, vestibular nerve and spinal cord. (*Schaller.*)

Tumors of the cerebellum are more often seen in children and they grow rather rapidly, so that a distinct picture of cerebellar disease may be developed in a few months. The early symptoms are vomiting, headache and choked disc; and the child may be first brought to the physician for stomach trouble. The stumbling ataxic gait then develops and with it a good deal of general muscular weakness or

parasthenia. Strabismus and nystagmus are often early symptoms. The patient has a weakness of one side not amounting to hemiplegia and there may be some facial nerve weakness on the same side as the lesion. There is diminution or loss of deep reflexes and some hypotonia. As the ataxia progresses, the patient may be unable to stand well, but inclines to fall backward or to one side; when he tries to sit, even, he may fall over and the hands become awkward and ataxic, though less so than the legs. There may be sudden seizures of violent vertigo, or loss of consciousness, with tonic irregular movements of the limbs, or perhaps only sudden short forced movements, throwing the patient to the floor (cerebellar seizures). These are less common in children than in adults. Other special symptoms of cerebellar disease elsewhere described may appear. These are nystagmus, jerky tremors of the hands, adiadokokinesis, asynergy in locomotion, dysmetria, cerebellar catalepsy and the disturbance of the normal labyrinthine reflexes as shown by Barany tests (see general diagnosis).

Fig. 221.—Attitude in a case of cerebellar tumor.

As the tumor grows it usually stops up the aqueduct from the third to the fourth ventricle, and then an internal hydrocephalus develops and we get symptoms of brain compression. The mind becomes apathetic and slow; headache and vomiting are less, blindness sets in, with paralysis of some of the motor nerves of the eye and dilated and fixed pupils. The neck is retracted and stiff and Kernig's sign is present. The pulse may be slow at first and later in the terminal stage may be signs of bulbar compression and paralysis. The patient emaciates and death occurs in one-half to two years; though in some slow-growing tumors of adults the disease may last ten years or more.

Tumors of the lateral lobes of the cerebellum should theoretically cause especially ataxia of the extremities, on the same side and a tendency

to fall to that side. Tumors of the vermis should cause more disturbance of equilibrium and cause a tendency to fall usually forward or backward.

Tumor of the ponto-cerebellar angle gives a rather special symptomatology. It arises usually as a small fibroma on the auditory nerve. It grows very slowly and may undergo finally sarcomatous degeneration. As it grows it forms a hard, circumscribed, pedunculated mass pressing into the angle between the lateral and anterior surface of the pons and the middle peduncle of the cerebellum. The first symptoms are simply those of irritation and compression of the eighth nerve, with tinnitus, vertigo, and progressive deafness; then there may be pains in the trigeminal nerve and later perhaps some paræsthesia or anæsthesia

Fig. 222.—Position of head in cerebellar tumor. (*Fraenkel.*)

over the distribution of this nerve with loss of conjunctival reflex. The facial nerve may become involved as the disease progresses. Usually not till after several years does the patient begin to show the general symptoms of brain tumor, and then those of cerebellar disease: headache, vomiting, choked disc, staggering, cerebellar gait, falling generally to the side of the lesion, cerebellar attitude. In about half the cases there are cerebellar seizures and there may be some hemiplegia on the side of the lesion; finally, symptoms of internal hydrocephalus and brain pressure appear.

Multiple Tumors.—About one-tenth of all cases of brain tumor are multiple. Hence, in making a diagnosis of the localization of tumors this fact must be borne in mind. The tumors which are most frequently multiple are tubercle, cancer, and melanotic growths.

Pathology.—Tubercle is the form of tumor found oftenest in children. It is more often located in the cerebellum, but may appear in the pons or other parts of the brain. It may be a single or, as it is then called, a solitary tubercle, or there may be a multiple growth. The tumor is irregularly round in shape and varies in diameter from one and a half to two inches. It has a grayish-yellow appearance externally; internally, a yellowish or cheesy look. It is not vascular, but is often surrounded by softened or inflamed tissue. There may be an associated meningitis. The tumors, when solitary, usually start from the central parts of the brain, but they also develop on the meninges of the convexity, particularly in the parietal region, and sometimes they develop also at the base. The tumors develop usually from some infectious focus, starting in a blood-vessel of the pia mater. Microscopically, the tumor shows the ordinary appearances of tuberculous growths. It contains in its periphery many round cells, nuclei, and giant cells. In the centre there is usually an amorphous substance, the product of degeneration and the breaking down of the ordinary substance of the tumor. The characteristics of the growths are the presence of the round cells and giant cells, the caseation and softening of the centre, and the absence of vascularization, with the presence of the bacilli.

Syphiloma or Gumma.—Gummatous tumors of the brain are usually associated with syphilitic meningitis and endarteritis and are usually found upon the brain surface, oftenest on the base, next upon the convexity of the frontal and central convolutions. The process appears either in the form of a somewhat distinct tumor or in the form of an irregular thickened exudate lying upon the surface of the brain and forming what is called gummy meningitis. The gummata may attain great size. They start usually from the pia mater and are due, as in the case of tubercle, to the irritative action of the infective organism. The gumma is irregular in shape; it has a somewhat thick, grayish periphery and often a yellowish centre, the appearances differing with the age of the tumor. Microscopically, it is found to consist of small round cells and spindle cells with various broken-down nerve-tissue elements. Gummata are at the present time very rare forms of brain tumor, as their development is usually controlled by treatment.

Actinomycosis is a form of infectious tumor which sometimes extends from the face and neck into the brain, leading to inflammatory processes, however, rather than to true tumors. No other neoplasms of infectious origin attack the brain unless sarcoma be found to be of that nature.

Cystic tumors are not uncommon and are the result of parasitic invasion, of injuries, and of the breaking down of a glioma or from a teratoma.

Glioma is the most frequent form of brain tumor. It may occur in any part of the brain, but is most frequently found in the cerebrum. It is the only tumor which is peculiar to the nervous centres, being developed from the neuroglia tissue which forms the supporting structure of these centres. Glioma originates in the white or gray matter of the nerve-centres and not from the membranes or fibrous structures. It may

Fig. 223.—Tumor of frontal lobe. (J. R. Hunt.)

grow to a very large size and is the form of brain tumor which becomes the largest. Gliomatous tumors measure from three to eight or more centimetres in diameter. In appearance the glioma can be scarcely distinguished from the brain substance itself, but usually looks like either pale or congested gray matter, or it may have a yellowish or gelatinous appearance. The tumor is very vascular and it may show the results of hemorrhages. The central part sometimes breaks down,

forming cavities or cysts. The tumor may grow very rapidly, infiltrating the normal tissue and in these cases there is hardly any definite boundary between it and the normal tissue. In other cases the tumor grows slowly, but rarely if ever becomes encapsuled. Microscopically, it is found to consist of small cells with delicate fibrous prolongations,

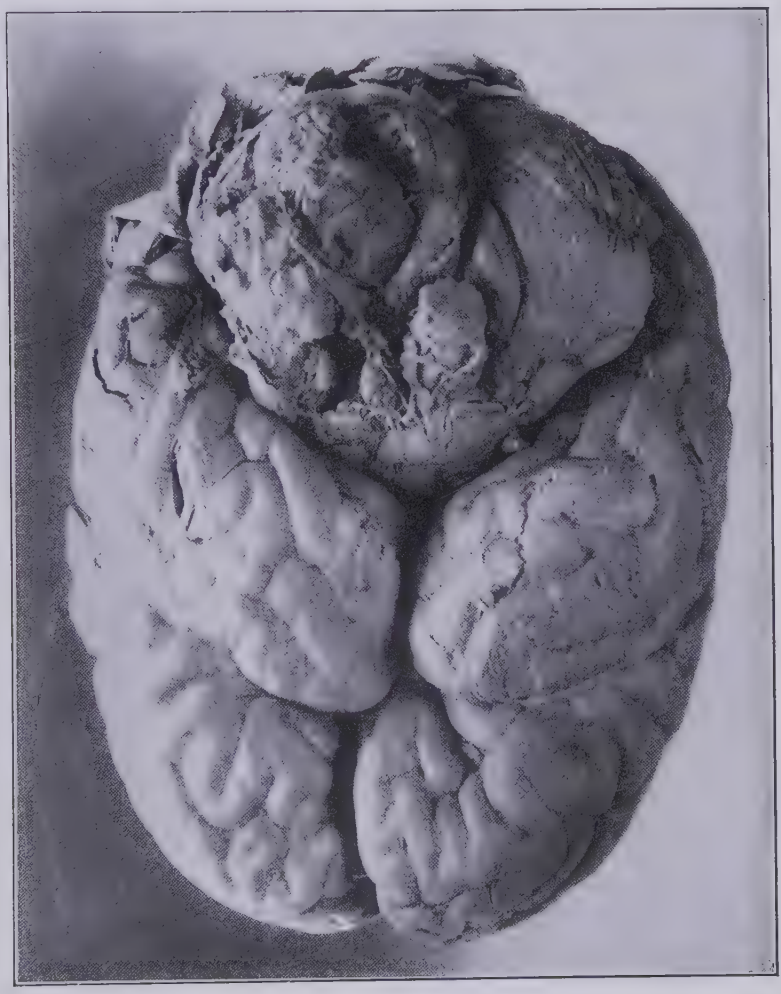


FIG. 224.—Tumor of cerebello-pontine angle.

these being the glia cells. Gliomata may undergo certain changes, *e.g.*, a mucous degeneration of the cells takes place, forming a myxoglioma. When there is with the neuroglia-cell proliferation a rich proliferation of round cells from the connective tissue, it is called a glio-

sarcoma. When the tumor is situated near the surface, involves the membranes, and grows slowly, with an increase in fibrous tissue, it is called a fibro-glioma. When the gliomatous growth is very firm and hard, the fibrous portion of the glia tissue predominates; it constitutes a nodule such as is found in multiple sclerosis, and these hard gliomata are sometimes called neuro-gliomata.

Sarcoma.—The sarcoma and its various modifications is next to glioma in frequency. It may be primary or secondary in origin. The sarcoma develops from the brain membranes or from the walls of the blood-vessels and nerve sheaths. Sarcomata may be single or multiple. They may be of all shapes and they grow often rapidly to very varying sizes. They often develop a capsule. They are white or grayish in appearance or may be somewhat yellowish, dependent on the predominance of the different kinds of cells and blood-vessels. Microscopically, they are made up of small round cells, spindle cells, and other cells of various sizes and forms. They contain often considerable fibrous tissue. They contain blood-vessels, but are not richly vascular. The essential characteristic of the sarcoma is the rich development of round cells and spindle cells; in other words, its rich cellular contents. Sarcomata are peculiar in undergoing many modifications. Thus sometimes fibrous tissue develops largely and the tumor is called a fibro-sarcoma; sometimes the tumor undergoes mucous degeneration and is called a myxo-sarcoma. There may be a breaking down of the centre with the formation of cysts. There may be a development of pigment. Not frequently a sarcomatous process invades a glioma and we have a mixture of a sarcoma and glioma. Sarcomatous tumors sometimes have an alveolar structure. These tumors contain endothelial cells derived from the lymphatics. When sarcomata develop from the dura mater and are slow in growth there may be calcareous deposits in them and they are called psammomata.

Endothelioma develops from the endothelium of the blood-vessels and lymphatic spaces. It usually starts from the meninges and is often encapsulated and therefore subject to surgical treatment.

The fibroma is a very rare brain tumor, unless the Pacchionian bodies, when enlarged and hardened, may be so considered. Occasionally fibro-neuromata are found developing on the roots of the cranial nerves.

Osteoma is not particularly rare, developing in the form of bony plates in the dura, falx, or tentorium. Osteomata in the brain substance are mere pathological curiosities.

Angiomata occasionally occur in the pia mater in connection with vascular nævi of the face, and also in the cortex and basal ganglia. They are also usually congenital.

Enchondromata and *lipomata* are extremely rare.

Cancer is relatively a rare affection of the brain, and is almost always of secondary origin.

Parasitic growths.—Parasitic tumors are extremely rare in this country. The forms which are found are the echinococcus and the cysticercus cellulosæ. The echinococcus produces hydatid cysts, which may be large or small, few or many, and are usually all upon the surface of the brain. They are much rarer than the cysticerci. These latter form cysts which are usually multiple, slow in growth, lie upon the surface of the brain or in the ventricles, are encapsuled, and show no symptoms.

Aneurisms are anatomically tumors, but clinically they present some special symptoms and hence are described separately.

Diagnosis.—It is necessary first to make the diagnosis of the presence of the tumor, next of its location, and finally of its nature. The existence of a brain tumor is determined by the presence of the characteristic general symptoms—headache, vomiting, vertigo, optic neuritis, mental disturbances, progressive course and local signs. The things which produce somewhat similar symptoms are localized meningitis (syphilitic or tubercular), brain abscess, localized foci of arterial sclerosis; lead poisoning, hysteria, acute internal hydrocephalus and certain toxæmias.

The physician must bear in mind the age of the patient and the existence of a tuberculous or syphilitic history, the history of an injury, of local tenderness, and the presence of some new growth in other parts of the body, particularly about the neck or thorax or in the lungs.

Blood tests assist in excluding inflammatory and luetic conditions. Lumbar puncture and examination of the cerebrospinal fluid helps to determine lues and meningitis; brain puncture and the withdrawal of a very small cylinder of brain tissue which may be examined microscopically has become a useful and safe operation. Percussion of the skull sometimes shows a duller note over the region of a tumor. Occasionally in young patients percussion brings out a cracked-pot sound. This only occurs when the intra-cranial pressure has loosened the sutures of the skull. In X-ray pictures one sometimes can see a darkened mass indicating the presence of a tumor. More often the X-ray only helps by showing changes in the bone, as in tumors of the sella turcica. Sometimes also in superficially placed tumors the plate shows broad lines indicating dilatations of the veins of the diploe.

The diagnosis of the location of the tumor is based upon the rules already given in regard to local diagnosis.

The diagnosis of the nature of the tumor can often be made and should be attempted. About two-thirds of tumors in adults are gliomata. Tubercle and glioma are the most common in children. Cerebellar tumors are often gliomata, especially in adults. Tumors of the ventricles are usually gliomata or adenomata. Tumors that develop without

many serious pressure symptoms are usually gliomata. Cancers are practically always secondary and sarcomata sometimes. Endothelioma are generally superficial, at first, starting from the meninges. The presence of syphilitic tumors can be inferred from laboratory tests. Sudden exacerbations of symptoms due to intra-neoplasmic hemorrhage suggest glioma.

Prognosis.—In extremely rare cases tumors of the brain appear to stop growing and become encapsulated and atrophied. Such tumors are of a tuberculous or syphilitic, perhaps sometimes of a gliomatous or sarcomatous, character. As a rule, the brain tumor grows steadily and the symptoms of the disease become more pronounced until death occurs. The prognosis is best for tubercle in children and gumma in adults. It is worse in cases of glioma and cancer. The disease lasts on an average two or three years, ranging from a month to eighteen years.

Treatment.—Something can be done medically in cases of tuberculous tumors, syphilitic tumors, and possibly in the sarcomatous variety. In tuberculous tumors a general constitutional and strengthening treatment must be resorted to; fresh air, rest, tonics, and proper food being the main reliance. In sarcoma and glioma, some help may be obtained from the internal use of arsenic. Iodide of potassium sometimes helps non-syphilitic tumors. Symptomatically we must give drugs for the relief of pain. The ice cap and leeching often help the headache. Should convulsions develop, the bromides should be used, just as in idiopathic epilepsy.

In cases in which the location of the tumor can be made out, the question of surgical interference should be considered. The percentage of cases in which surgery can effectually help is small. It will include only those cases in which the tumor can be located; of those which can be located, only those which are in an accessible region, and finally, of those which are in an accessible region, it includes those whose removal will not leave the patient helpless, demented or aphasic. The removable tumors of the brain amount to less than 5 per cent. They are, in particular, the endotheliomata, sarcomata and gliomata. These latter sometimes degenerate and become absorbed after partial removal. Tumors can be removed with possible benefit from the superficial part of any portion of the cerebral hemispheres except the left temporal lobe. Here the patient if operated on would be made aphasic and probably hemiplegic. Tumors can be removed from the cerebellum and the cerebellopontine angle with occasional success and even with brilliant results. Tumors can be removed from the hypophysis, by the very expert. Tumors of the mid-brain, pons, medulla, and of the ventricles are not operable. In cases which are not operable, the decompressive operation of Cushing or puncture of the corpus callosum may often be done with advantage.

Lumbar puncture can be employed to test and to a slight extent relieve the intra-cranial pressure. The measure should not be employed, however, in subtentorial tumors as the medulla may be pushed into the foramen magnum and death ensue. The ordinary pressure of the cerebrospinal fluid of 40 to 150 mm. of water may in brain tumor be increased to 250 and 900.

INTRA-CRANIAL ANEURISMS

Intra-cranial aneurisms are of two kinds—"miliary" and those of large size. The miliary aneurisms are minute dilatations of the vessels and are always multiple; they have been described under the head of cerebral hemorrhage. Large aneurisms affect only the large cerebral arteries at the base of the brain. The arteries are affected in the following order: middle cerebral, basilar, internal carotid, and anterior cerebral. The anterior and posterior communicating and vertebral arteries are occasionally involved, the posterior cerebral and inferior cerebellar very rarely (Gowers).

Etiology.—Males are affected slightly oftener than females. Aneurisms occur at all ages from ten to sixty; before ten and after sixty they are extremely rare. Heredity occasionally plays a part in predisposing to cerebral aneurisms. The exciting causes are embolism, especially when the emboli contain microbes, syphilitic disease, injuries, and in rare cases senile arterial degeneration.

The *symptoms* are very indefinite; they resemble to a considerable extent those of tumor at the base of the brain; headache and vertigo, mental dullness and irritation, cranial-nerve palsies, and occasionally hemiplegia and convulsions are noted. Optic neuritis is rather rare. In a few cases the patient is conscious of a murmur or recognizes the pulsating sensation in the head. Sometimes when the aneurism is in the vertebral artery, a murmur can be heard between the mastoid process and the spinal column (Moser).

The *diagnosis* is often difficult; it is based on symptoms of tumor at the base of the brain pressing on cranial nerves and on motor or sensory tracts. The effect of carotid compression should be tried.

The *prognosis* is not good. In perhaps the majority of cases a rupture of the vessel occurs in a few years; however, rupture is not the inevitable event, and sometimes the disease becomes stationary or undergoes spontaneous cure.

The *treatment* of the disease, if it can be recognized, is the same as that for aneurism elsewhere, so far as drugs are concerned. The use of salvarsan has produced good results; surgically, the common carotid may be tied (though this involves some risk) and perhaps the vertebral if the aneurism is believed to be connected with that artery or with the basilar.

CHAPTER XX

FUNCTIONAL AND DEGENERATIVE DISEASES—EPILEPSY

Functional is a term applied to nervous diseases in which no known anatomical change underlies the morbid phenomena. Degenerative diseases are those due to or associated with a constitutional deterioration, or biogenetic defect. The number of diseases of functional character is steadily lessening, and the use of the term, therefore, is being supplanted by other qualifying words.

Epilepsy is a disease of the functional and degenerative type, although the essential and dominant symptoms are due to various morbid conditions. It is a chronic nervous disorder characterized by periodical seizures attended by loss of consciousness and usually by convulsions. Mental disturbances may accompany or take the place of the convulsions. The mental make-up of the patient is generally peculiar and if the disease progresses some mental deterioration sets in. It has no established pathological anatomy, although there are degenerative cell changes in the brain cortex.

Symptomatic epilepsy is a form in which the periodic convulsive attacks are associated with gross organic changes in the brain, or is the expression of a definite epileptogenous irritation (toxic, vascular, psychic or reflex).

Jacksonian or *partial epilepsy* is a form of epliepsy, usually symptomatic, and is characterized by periodic convulsions affecting only certain groups of muscles, and often unattended by loss of consciousness.

Hystero-epilepsy is not epilepsy, but a form of hysteria.

Eclampsia or acute epilepsy is the name given to a single isolated attack of convulsions. It is generally of the symptomatic type.

Ordinary or "idiopathic" epilepsy shows itself in three rather distinct types of attacks, viz., that of severe attacks called the *grand mal;* that of minor attacks, the *petit mal;* and the rare larvated forms characterized by acute mental disorder and called *psychical epilepsy* or the psychical epileptic equivalent.

Etiology.—Predisposing causes: Heredity is the most potent of any single influence. A history of epilepsy, insanity or some serious neuropathic condition is found in the family in over one-third of the cases and rather more on the paternal side.

In many other cases it can only be established that the family is a neuropathic one. The heredity is rarely direct; *i.e.*, the parents are not epileptic in more than about 1 per cent. of cases. Alcoholism and the intermarriage of neurotic persons contribute to produce the convulsive tendency in children. The element of alcoholism, in my experience (private and public) is only about 5 per cent. It must be remembered that many parents reported alcoholic were not so at the time of the birth of the child. Powerful emotions during pregnancy, accouchement injuries, and syphilis, have slight influence. The Wassermann test for syphilis in epileptics has brought out a small percentage of positive reactions. Very important causes of epilepsy are cerebral or meningeal hemorrhage and attacks of encephalitis occurring in early life. Such attacks may lead to mental defects and paralysis; about 40 per cent. of these cases have epilepsy.

More cases occur in the country than the city, more in temperate climates, and more among in-bred races. American statistics show a slight preponderance among males. The epileptic age is between birth and twenty, and still more definitely between five and fifteen. In three-fourths of the cases the disease begins before the age of twenty; in one-sixth of my cases, before the age of five. After twenty the danger of epilepsy is slight, and when it occurs it is usually due to accidental causes, like syphilis, alcoholism, or plumb ism. Idopathic epilepsy, however, may develop even after sixty. The accompanying table shows graphically the relation of age to the development of epilepsy, chorea, and neuralgias.

Exciting causes are not present in the majority of cases. The most important are the occurrence of rickets at the time of dentition, injury to the head, sunstroke, acute infectious diseases and toxæmias, persistent reflex irritations and alcoholism. The importance of traumatism when there has been a real injury to the brain is considerable. Simple concussions without organic injury do little harm. Masturbation is a real but rare exciting cause. The so-called reflex causes are ocular and auditory irritations, worms, gastro-intestinal weakness, dental irritations, and lesions involving peripheral nerves. Probably the gastro-intestinal tract and genital organs furnish the most important exciting irritations.

X-ray examination often shows some defects in the position and functioning of the digestive tube with ptosis, kinks and a patent ileocæcal valve.

A chronic epilepsy may be brought out by peripheral irritations while more rarely there occurs only a reflex epileptiform neurosis, the convulsions subsiding when the irritation is removed.

Epileptic seizures of characteristic type may be caused by psychic

states. In other words, an apparent epilepsy may be psychogenous. Some followers of psychoanalysis claim that the convulsive seizure itself is the expression of a suppressed wish or instinct of infantile origin.

Symptoms.—*The Onset.*—The disease begins sometimes with attacks of *petit mal* which may last one or more years before anything more serious occurs. Then suddenly there is a convulsion. Occasionally nothing but *petit mal* attacks ever occur. In other cases without previous warning the patient has a severe seizure. In six months or a year he has another, in the next year he has three or four and then they come on regularly every week or two.

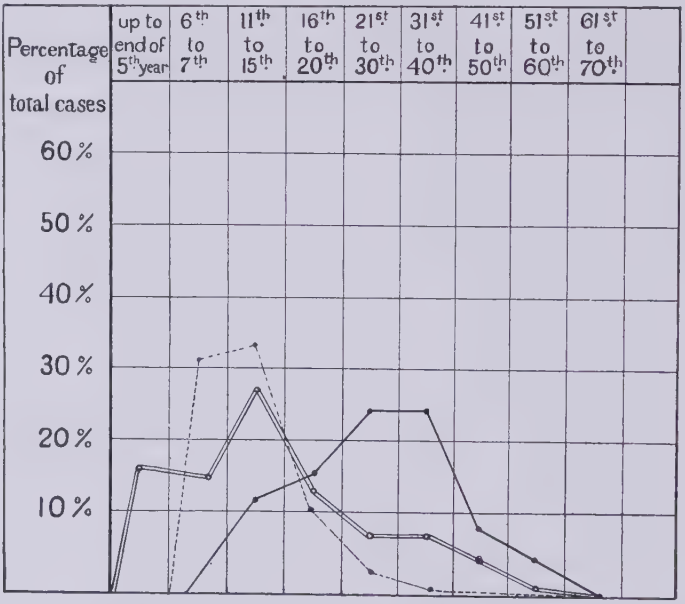


Fig. 225.—Table showing percentage of cases of epilepsy, chorea, and neuralgia occurring at each half-decade and (after twenty) each decade. Double line, epilepsy; dotted line, chorea; single line, nuralgia.

The Convulsion.—The patient often feels some premonitory symptoms for a few hours or a day, consisting of general malaise, irritability, or giddiness. The attack begins in about half the cases with a peculiar sensation called the aura. Often also a loud cry is uttered and the patient falls unconscious to the ground. The face is pale, the eyes are open and turned up or to one side, and the pupils dilated. The head is drawn back or to one side, and the whole body is in a state of rigidity or tonic spasm. The arms are slightly drawn out from the trunk, the forearms and wrists extended or flexed, the fingers clinched or flexed in various ways, the legs and feet extended. This tonic stage lasts for fifteen

or twenty seconds; the face becomes congested and then livid from compression of the veins of the neck and stoppage of respiration. Gradually jerky movements of the face and limbs begin and the stage of clonic spasm sets in. The trunk and limbs are now alternately flexed and extended with violent shock-like contractions, the facial and eye-muscles twitch, saliva collects in the mouth, and as the tongue is often bitten it becomes stained with blood. The movements are sometimes so violent that the patient is thrown about the bed or floor, and occasionally a limb is dislocated, usually the shoulder. The urine often, and the fæces occasionally are passed. The temperature is raised ½° or 1° F., rarely more. The pulse, feeble at first, becomes frequent and tense, and then, as the attack subsides, becomes feeble again. The clonic spasm lasts from one-half to one or two minutes. It subsides gradually, and the patient sinks into a stupor, from which he can be roused with difficulty. This stupor is succeeded by a heavy sleep of several hours and a feeling of hebetude which lasts all day. Vomiting sometimes occurs as a terminal symptom. Immediately after the attack there is a temporary exhaustive paralysis, with loss of knee-jerk. The pupils contract again and often oscillate. There may be a slight amount of transient albuminuria or glycosuria. The earthy phosphates are found increased; urea is not. There is a distinct lessening of hæmoglobin in the blood (Féré) and of hæmatoblasts. Sometimes the attack is followed by others, and for hours the patient passes from one convulsion into another. This condition is called *status epilepticus*. It usually lasts less than twelve hours, but may last for one or more days and until finally death occurs from exhaustion. It develops only in the severer types.

Symptoms of the Minor Attacks.—In the minor attacks (*petit mal*) the patient suddenly stops in anything in which he is engaged, the features become fixed, the eyes are open, the face is pale, the pupils are dilated, often slight twitching of the facial muscles or of the limbs occurs, and consciousness is lost. In a few seconds the attack is over, and the patient, who does not fall, resumes his work or conversation, being unconscious of what has occurred, except that he has had a "spell." Often there is a warning sensation of aura. This is felt as giddiness, sense of fear, numb sensations of extremities, flashes of light or blindness, or choking sensations. There may be a cry uttered. The minor attacks are in rarer cases accompanied by sudden forced movements; the patient runs a few steps; or turns round, or makes some automatic movements. This is called *procursive epilepsy*. Sometimes the patient suddenly falls to the ground, as if the legs had given way. He immediately rises and is quite normal again. There is no vertigo or apparent loss of consciousness. This is a true "falling sickness." Sometimes he makes

simply a sudden very violent nodding movement of the head. This is the epileptic "salaam spasm."

Symptoms of the Psychical Attacks.—Sometimes the minor attacks are followed by outbursts of maniacal excitement or by sudden violent automatic movements, and in these states the patient may commit crimes of violence. In rare cases the patient passes into a somnambulic state, during which he performs accustomed acts, such as driving and walking, automatically and naturally (somnambulic epilepsy). This form of epilepsy may come on without a preliminary minor attack, and then it is to be considered a "psychical epileptic equivalent."

Minor attacks may end in convulsions of a co-ordinate type in which the patient jumps, kicks, throws the arms about as in hysterical attacks. These are called hysteroid convulsions.

The seizure may consist of only a short tonic stage and a few twitchings of the limbs, the whole lasting but a few seconds. This is called an *abortive attack.* Under the influence of medication, the severe seizures are often reduced to abortive forms.

Jacksonian or *partial epilepsy* is a form of the disease characterized by convulsive attacks affecting only a single group of muscles or a limb, and generally not accompanied by loss of consciousness. Jacksonian epilepsy is always symptomatic of some focal lesion affecting the cortical motor area of the brain. This may be a tumor, inflammation, or injury, or only some slight degenerative change in the cells of the cortex. This form of seizure is particularly significant of a slowly growing brain tumor or syphilis.

The *epileptic aura* usually consists of a sensation of numbness, prickling or of a breeze beginning in the hand or leg and passing up to the head when consciousness is lost. Still oftener there is a peculiar sensation starting in the epigastrium and passing upward. More rarely there are special-sense auræ, such as a ball of light in one eye, noises, or voices or peculiar tastes or smells. Besides these there occur sudden desire to go to stool, feelings of giddiness, dreamy states, peculiar sensations in the head, and indescribable general sensations.

The auræ may be divided into:

Visceral—epigastric, laryngeal, cardiac, rectal.

Cutaneous sensations.

Special senses—flashes of light, etc.

Psychical—emotions, dreamy states, etc.

Cephalic—giddiness, etc.

The aura is thought to indicate the seat of the first discharge of nerve force, and its study is of most importance in connection with symptomatic epilepsies, as will be shown later. The patient may have only the aura for a long time before the convulsion sets in. Sometimes there

never is anything but the *aura*. This undeveloped form of epilepsy I have called *paraepilepsy*.

Frequency of the Attacks.—Epilepsy with the severe attacks is the most frequent type, next come combinations of severe and minor attacks, and next minor attacks alone, while the psychical forms are the rarest. The severe attacks may come on only once or twice a year, and this commonly occurs during the development of the disease. The frequency gradually increases until they occur every month, or two or three times a month. Sometimes the fits occur in groups of four or five every month or two. In very bad cases convulsions occur every day. The *petit-mal* attacks are more numerous and often occur daily.

Time of Attacks.—The moon and the seasons have no influence. More attacks occur during waking hours than during sleep; but two-thirds of the attacks occur between 8 A.M. and 9 P.M. Many patients have their attacks early in the morning just after awakening (*matutinal epilepsy*). According to Pierce Clark, the hour in which the greatest number of attacks occur is 9 P.M., and the fewest attacks occur between 4 and 7 P.M.

State of Patient Between Attacks.—Epileptic patients often feel better for a time after the convulsion is over. After very severe attacks or a series of them there may be a clouded consciousness, amnesia and mental confusion for several days. They not rarely suffer from severe neuralgic headaches; the appetite is capricious, often in children it is voracious, but in older cases there may be anorexia; the bowels are usually constipated; the pulse is small, soft, and frequent in the young, later it is often slow. The blood pressure is usually low.

Mental Condition.—A gradual mental deterioration occurs in the great majority of epileptics, but it is slight in some and not very serious in others. It shows itself by feebleness of memory, irritability of temper, selfishness, incapacity to concentrate the mind or to carry out a purpose. In children great mischievousness and lack of moral sense, with vicious, impulses, may appear. The mental deterioration is dependent on those underlying factors which cause the disease. It is apparently associated with the excessive number of the fits, and the accompanying degenerative changes in the cortex of the brain. This is not always the case, nor is it generally true that deterioration occurs more often with *petit mal*. It is more marked in cases beginning very early in life, but this is true only when there are early and decided marks of physical and mental degeneration present. A certain rather small percentage of epileptics become either demented or insane. True epilepsy is rarely compatible with extraordinary intellectual endowments. Cæsar, Napoleon, Peter the Great, and other geniuses may have had symptomatic fits, but not epilepsy.

Physical Condition.—Epileptics are usually undersized and of not very robust constitution (Féré). Some patients, however, present a very vigorous muscular development. They always present some of the marks of degeneration, physical, physiological, or mental. Such marks or stigmata are about ten times more frequent than in healthy persons. The physical stigmata are (Féré) short stature, cranial asymmetry (in 71 per cent.), short parietal or frontal arc, and triangular skull; in women high prominent forehead; bad teeth badly placed, high palatal arch; facial asymmetry; prominence of occiput and lemurian hypophysis; differences in color, size, position and shape of pupils; astigmatism (in 75 per cent. of cases); badly shaped and placed ears; misplaced crown of scalp; low vital capacity; small genitals, atrophic uterus; greater development of left side; long fingers. Cranial deformities of pronounced type occur in epileptics associated with idiocy, hemiplegia and brain defects of early origin. Sometimes, apparently from a premature ossification of sutures, there are the peculiar shapes of the skull known as scaphocephaly, or steeple skull, and plagiocephaly, or obliquely deformed skull.

The physiological marks of deterioration are a lessened muscular strength (as 35 to 50), habit choreas, a rather imperfect vision with excessive amount of astigmatism and functional muscular weakness. There is a lessened vital capacity, weak and slow digestion, often defective metabolism and sexual atrophy or irritability.

The psychical stigmata are mental feebleness, moral insensibility, a certain unreasonableness, lack of insight and irritability. Some have wayward and vicious impulses, lack of will power and sexual aberrations.

There are cases of epilepsy, however, in which the mental powers are fairly normal and continue well preserved; and I am describing the non-custodial, non-demented cases.

Association Diseases.—Epilepsy may be complicated with a degenerative myoclonia (myoclonus epilepsy). It is sometimes seen in connection with torticollis and spasmodic tic of other types. Epileptic seizures occur in cerebral syphilis and general paresis.

Epilepsy is regarded by some as not a disease, but a syndrome or symptom-complex due to various morbid conditions of the brain. It is true that epilepsy runs a varying course and presents different features in different individuals. The grouping of cases in accordance with the severity and type of attack, the degree of intelligence, the tendency toward deterioration, the physical and physiological defects, the tendency to arrest improvement, or increased severity of the disease is, therefore, desirable. Taking into account these factors, I find in my experience the following general types or groups. It is not pretended that this is an exact classification. Such a one is not yet possible. It is fairly accurate,

as I have seen epilepsy and I have watched many cases for ten to twenty-five years.

The types are:

I. *The Common Mixed Form.*—The attacks begin about puberty. They are of *haut-* and *petit-mal* type, but it is the *haut-mal* that is troublesome. The seizures settle down to four to six monthly and occur singly or in groups, by day or night.

The patients have some morbid mental traits; they are of moderate intelligence and this deteriorates a little, but does not end in insanity or dementia. The physique is fairly good, often robust, as to the muscular system; there are generally defects of physiological function. These patients sometimes get well or get their attacks well under control.

II. *The Petit-mal and Psychic Type.*—The attacks are of the psychic and mild type alone, at least for very many years. They come on, as a rule, in early childhood at the fourth and fifth year or even sooner. They increase until they occur many times daily. The patients are bright, often very bright and precocious children, and they do not dement as the years go on, except very late or unless *haut-mal* attacks set in. They may, however, develop mental peculiarities and defects. Physically, they are of delicate but sound physique. These cases do not get well. With this *petit-mal* type go most of the cases of cerebral automatism, psychical epilepsy, epileptic sleep attacks, etc.

III. *The Delayed Type, Epilepsia Tardiva.*— The attacks do not come on until after adolescence, and sometimes not till the climacteric. They are of *haut-mal* type, but *petit mal* is usually present in some degree. There is sometimes a history of cause, such as infection, trauma, syphilis, overstrain and arterial sclerosis. The attacks are not very frequent relatively, *i.e.*, one or two a month. They are controlled fairly easily, though not entirely. The patients are of fair intelligence, of higher degree at least than those of type I. They do not deteriorate very much. The epilepsy of advanced years is not of this type, but is a phase of senile dementia.

IV. *The Organic and Degenerate Type.*—The attacks come on in connection with gross defects or disease or injuries of the brain. There is retarded development, idiocy, imbecility, perversions and lack of balance in the mental functions. Paralyses, tics, etc., are present. Here the epilepsy is only one expression of the morbid cerebral condition. The cases are hopeless, so far as cure is concerned.

Underlying all the types there is, as a cause of the seizure, some psychic state or degenerative neuron change. It is very probable that this change is of very much the same character in all cases, and that the disease differs in course and symptoms because the changes vary in locality, extent and intensity. Even in idiots and paralytics the epileptic phenomena are

due to, or associated with, this special finer cellular degeneration, for the brain may be greatly injured, badly developed, full of cysts or tumors and disease, yet the epilepsy does not always appear.

V. *Psychogenous Epilepsy.*—There is a disease in which the patient suffers from apparently true epileptic convulsion, of *grand-mal* type. Psychoanalytic and clinical study, however, shows that the convulsions are of psychic origin, due to some irritating mental state. The convulsion is thought to be the result of this disturbing state.

Pathology.—The body of the epileptic shows sometimes skin eruptions and ulcers, the result of treatment. There are often evidences of local injuries and fractures due to falls. The organs may show vices of conformation. The uterus is frequently infantile or sharply flexed. Deformation of the occipital bone or the atlas so as to produce narrowing of the upper spinal canal has been noticed. The brain may be unduly large or small, but there is nothing constant in this, nor is there an abnormal difference in the weight of the two hemispheres. The convolutions show many anomalies, but there is in them nothing specific. On the whole the convolutional type is a simple one. The pathological change found in more or less advanced epilepsy is a progressive gliosis with cell degeneration in the cortex and basal ganglia, and the cornu ammonis. Turner finds some characteristic changes in the cells of Betz and blood-vessels. In old cases there is often a chronic leptomeningitis, and vascular changes due to the frequent congestions of the brain take place. These consist in varicose and fusiform dilatation of vessels with evidence of small hemorrhages. Slight degenerative changes in the nerve-fibres are also observed. While cortical degeneration and gliotic foci are formed in epilepsy, they are the expression of some pathogenic state not the cause of it.

The epileptic fit itself is a special manifestation of the disease which may be of toxic or bio-chemical of psychic origin. It is not likely, however, that the psychic factor can do its work on any but a psychopathic brain. It is probable that in true epilepsy there is an instability of certain cell layers of the cortex cerebri, and a tendency to degenerate. Whether this is due to a congenital weakness, or the action of endogenous poisons, or both we do not yet know. The many elaborate investigations that have been made to ascertain whether epilepsy is not due to some toxic cause or perverted metabolism or endogenous ferment, have not established anything. Even in alcoholic and lead epilepsy, the toxic agent is probably a secondary one.

The diagnosis is based on the character of the attacks and has to be made from hysterical and various toxic and symptomatic convulsions.

The aura, the scream, the quick loss of consciousness, the dilated, pupils, the tonic convulsion, the bitten tongue, the emptied bladder,

temporary loss of reflexes are all characteristic. The hysterical patient sometimes, but rarely, loses complete consciousness, the epileptic almost always. Hysterical patients do not hurt themselves in falling or bite their tongue, and their muscular movements, while irregular and violent in character, are yet co-ordinate, *i.e.*, they throw themselves about, kick, strike, etc. Their attacks often are produced by emotion and ended by some powerful mental or physical impression. They rarely occur in sleep. The slight rise of temperature in epileptics rarely occurs in hysterics. *Petit mal* and epileptic vertigo are distinguished by the sudden lapse of consciousness and by the sudden pallor and fixation of the eyes, dilatation of the pupils, and slight twitchings of the face. Nocturnal convulsions are usually epileptic.

Eclampsia, or acute symptomatic and reflex convulsions, cannot always be distinguished from epilepsy. The history of the case, the irregular and often prolonged character of the fit, may enable one to make the diagnosis.

Course and Prognosis.—Epilepsy shortens life to some extent; most subjects do not live beyond the age of forty or fifty. About 10 per cent. become demented or insane. Five or ten per cent. get well. The remainder reach a certain stage of severity in their disease and continue in it for years. This severity depends on the treatment, the nature of the attacks, and the extent of degeneration which the organism shows. While unquestionably treatment cures or suppresses the disease in some cases, it disappears spontaneously in others. Epilepsy developing after adolescence is not easily curable, but is rather easily controlled and is not so serious, except sometimes in old people. The prognosis of *petit mal* is worse than that of *grand mal;* that of the two combined is worse still, yet not hopeless. The psychical form of epilepsy is the least amenable to treatment. Epileptic insanity and dementia are incurable. Death occurs rarely in the attacks except in terminal stages. Yet the *status epilepticus* is always a source of danger.

Epileptics who have only a moderate number of attacks six to fifteen yearly, can get along comfortably for years, doing their work and enjoying a fair share of the duties and pleasures of life.

Treatment.—The first and essential rule of treatment is to take cases early and treat them vigorously from the start. Children who have had a few convulsions during the first three or five years of life should be treated as if they might develop epilepsy between the ages of ten and fifteen or earlier. The recurrence of a fit between the ages of five and ten should excite apprehension and call for the most diligent treatment. Another rule is that when epilepsy is recognized in children the case should be treated constantly for at least three years after all attacks have ceased.

Constitutional Treatment.—We should use measures that increase vasomotor tone and strengthen and steady the circulation. Nothing does this better than water. Epileptics should be given showers, douches, cold sponge baths, or wet packs according to their needs and opportunities. They should also drink water freely. Again, the nervous system is greatly steadied and quieted by mental occupation that interests one. Nothing is more unfortunate than the idleness often enforced on epileptics. I have seen the disease absolutely checked by having a boy learn a trade that he liked.

A second important indication is diet, and the prevention of intestinal decomposition. In *petit mal* particularly an absolutely non-irritating diet, such as milk, fruit and bread, will quickly lessen or stop the attacks. Meats can be taken in moderation if eaten slowly. As a rule it is a little safer to keep meat out of children's diet for a time; but in adults it is not necessary, though it should be given in moderation. A practically salt-free diet is advisable part of the time.

Removal of Irritating Causes. The rheumatic, gouty, and so-called tuberculous diatheses do not stand in any close relation to epilepsy. The condition known as lithæmia, however, in which there are insufficient oxidation and excretion of products of tissue waste, needs attention. Hence the use of bicarbonate of potassium, salicylate of sodium, the alkaline mineral waters, and a restricted diet are not rarely indicated.

The importance of reflex irritations has been much over-estimated. Still they must be considered. The most serious are those arising from the gastro-intestinal tract, the sexual organs and the eyes. Phimosis if present must be relieved, and masturbation or sexual excesses stopped, if possible. It is admitted now that removal of the ovaries, even if diseased, never cures true epilepsy, though it may help hysterical convulsions.

Astigmatism and hypermetropia should be corrected; also ocular insufficiencies if these are pronounced.

Proper attention to the frequent constipation and dyspepsia is of course necessary.

Specific Treatment.—Physicians who undertake the treatment of epileptics often do not realize the seriousness of their responsibilities. Many, I fear, simply give a little bromide, stop the meat, circumcise the boy, and say they think the child will outgrow it. But children do not outgrow it; they steadily get worse unless something definite is done, and well done for a long time. With proper, prompt, and prolonged treatment, the attacks can be entirely controlled in 5 to 10 per cent. of cases, and I believe more. They can be greatly controlled in over one-half the cases, so that the patient may be able to continue his education and do some work in life. But this cannot be accomplished

by any casual dosing or occasional consultation with some high authority. The physician should approach the responsibility of a case of epilepsy as he would that of a mortal surgical condition, in which much depends on knowledge and attention to all the details of a long technic.

It is conceded that the colony treatment of epilepsy is the one which approaches most nearly the ideal in effectiveness, but it cannot be applied to all classes—at least, for a long time—and perhaps never to a certain rather large percentage. There must always, therefore, be a good many epileptics who have to be treated at their homes, and whose care must be directed by the family physician or the specialist.

For this class of persons I have gradually evolved a somewhat specific treatment of epilepsy, which seems to produce the most satisfactory results in those cases in which a reasonable opportunity for therapeutic effort exists. That is to say, cases which are not of very long standing, and which have not already undergone serious mental deterioration, and cases in which the mental and physical degeneration, at the beginning, is not of a very high grade.

The features upon which emphasis must be laid, in the treatment of epilepsy, are:

First, the fact that the course of treatment about to be instituted is to last for at least two years, and that all measures prescribed must be carried out with the greatest fidelity and exactness during that time, no matter how well the patient may seem, or how unnecessary regimen and drugs may appear to be. The preparation and outlining of treatment should receive the care and attention such as is given to a capital operation.

Second, the use of the pure bromide of sodium or some other salt (there is no special merit in mixed bromides) in combination with the glycero-phosphate of soda or of soda and lime so that a patient takes on an average 60 grains of the bromide of soda and 20 to 30 grains of the phosphate, in twenty-four hours. To this combination iron and a little arsenic may be added at times. By the combination of proper soluble phosphates with bromide and eliminative measures, the depressing effect of the bromide can be greatly lessened and the patient can continue bright and active under a fairly large dose of the drug. This has been tested by me now for fifteen years, mainly in private practice, but also in dispensary work. The maximum dose of bromide which can be taken in this combination is sometimes as high as 90 grains a day, but rarely more, and not often as much. The phosphate should occasionally be omitted, and the form changed. Certain patients cannot take bromides, however, and are not benefited by them in any way.

Third, I have found it often an efficacious plan in treatment to intermit the medication for either one or two days in each seven. During

these days, *e.g.*, a Wednesday and Sunday, the drug is stopped, and in its place is given, three times a day, before meals, a tumbler of hot water, and with it an alkaline laxative. The ordinary tablet of rhubarb and soda, with nux vomica, usually answers this purpose. Sometimes 20 grains of bicarbonate of soda are enough. Its purpose is to flush out the stomach and bowel and cleanse the gastro-intestinal canal twice a week, thus preventing the accumulation of drugs and toxins. After meals on these days I sometimes give 10 drops of tincture of iron or some needed tonic. I have not seen any access of convulsion during the day or the day after such intermission. With this stomach treatment there should be in many cases a colon irrigation using a hot high bicarbonate of soda solution. This is given about once a week. Sometimes in its place I use an injection of a pint of warm sweet oil at night followed by a simple enema in the morning.

Fourth, an important measure which I employ in the treatment is the securing of active physical exercises for about twenty or thirty minutes, at least, three times a week. This must be done either by some active sport, like boxing, by exercises in the gymnasium, by tennis, skating, or by the simpler methods of chopping or sawing wood skipping the rope, dancing, or punching the bag; but the exercises of whatever kind should be short and to the point of free perspiration. After this exercise, the patient is given a cool bath. Delicate persons, women, and those who are unable to carry out such exertion, I direct to purchase a "hot-box" and take a hot-box sweat, followed by a cool bath, three times a week. These boxes are not expensive, and can be set up with little trouble.

Fifth, the question of diet is attended to, as indicated above.

The medication should be *increased* rather than diminished as the patient gets better; he should take more, if possible, at the end of the second year than at the beginning of the first, if the attacks are controlled. After four years one can feel safe, not before. There are few other drugs of any importance in epilepsy, though freak cures happen under all kinds of measures. Tincture of simulo seems to have some value in doses of ℥i. ter in die upward. Atropine in very large doses (gr. $\frac{1}{20}$ to $\frac{1}{75}$) gradually increased sometimes helps *petit-mal* types strikingly. Veronal and luminal may sometimes be given in cases in which bromides have no effect, or to aid their effects.

There are some cases in which a glandular defect exists, hypothyroidism being the most frequent in my experience. Small doses of thyroid, gr. $\frac{1}{4}$ to gr. ii., may be indirectly helpful. The other glands may be used as indicated. They only act as adjuncts.

Growing children must, as a rule, be taken from school. This is unfortunate, but it seems indisputably necessary. The education, however,

should be allowed to proceed and regular fixed short hours of study be given. The child should be allowed to live as nearly a normal life as possible. If old enough, some can be taught a trade or some handicraft with advantage. The grown person should be urged to adopt or continue in some occupation, of which an out-door one is preferable, but any occupation is preferable to idleness.

The most difficult cases after the degenerates and those with organic brain disease are the bright, precocious children who develop *petit mal* alone and who have daily attacks. They are usually not amenable to bromide medication in any form. After a thorough trial of this, however, even to the point of bromidation, and using every possible tonic and eliminant, the patient is best left to hygienic measures alone.

This treatment of epilepsy always presupposes a preliminary elimination of all reflex causes. If the epilepsy is local (*i.e.*, Jacksonian) or even if it began as a local affair, it is advisable to operate, and if any source of irritation is seen in the cortex it should be removed. Occasionally this does good, but not always. Often epileptics are brought to us with scars on the scalp and a history of some old injury, perhaps even of fracture. If the epilepsy is general, however, and does not seem to have started from the injured zone, operations are of no use.

It goes without saying that there are many epileptics who are too advanced in the disease or too degenerate for treatment and who need only custodial measures.

CHAPTER XXI

THE PSYCHO-NEUROSES

CONSTITUTIONAL INFERIORITY, HYSTERIA AND ALLIED STATES, PHYCHASTHENIA

Introduction.—The term psycho-neurosis is given to a number of disorders in which the nervous symptoms are in the main dominated or caused by the mental state. This mental state is a morbid one, but is not of sufficient degree to call for custodial care or justify applying to it the term insanity. It is just a minor psychosis.

There is a difference of view, as to the grouping of the psycho-neuroses. One has to follow some practical method, and I include under this term; (1) The psychopathic diathesis or constitutional inferiority. (2) Hysteria and allied states. (3) Psychasthenia. (4) Certain mild and abortive forms of the major psychoses.

The study of the pathogenesis of the psycho-neuroses takes one into difficult and unsettled fields, but the situation as between clinical and dynamic neurology may be stated as follows:

The clinician finds that an apparently healthy person has gradually or suddenly developed an automatic state, a fixed idea, or a functional paralysis. He inquires into the causes and finds (*a*) a nervous heredity (*e.g.*, the father alcoholic, the mother tuberculous); (*b*) the patient herself of nervous type and (*c*) a sufferer from an emotional strain or shock followed by the psycho-neurosis. (*d*) He learns the patient's mental make-up and intellectual equipment, the temperament and the bodily condition.

The data gained under *a*, *b*, *c*, *d*, tell him the cause of the disorder and show along what lines to work for cure. This is descriptive or clinical neurology.

But the dynamic neurologist goes further and searches into the intimate mental mechanism of the process by which the mind became thus morbidly changed. This mechanism according to some consists in a blocking of association processes and a limiting and re-grouping of neural units.

Thus the phenomenon of double personality, hypnotism, hysterical crises, trance, are explained, if it is an explanation, by supposing that there is a break and re-grouping of the association-work of the brain. Ordinarily the associations aroused by the stimuli of thought, emotion or sense have free play in accordance with what the individual's environment and training have developed. If he is hypnotized or obsessed this free play

of association does not take place. The patient hears and sees and thinks only what is suggested to him, by the hypnotizer or by his dominating idea. All his mental activity is along a particular group of associations. At the synapses of the nerve cells, there is a blocking off of the ordinary to-and-fro play of the neural currents. This same process occurs when the personality becomes changed. One neural unit of association activities underlies the personality of Mr. Hyde and another group those of Dr. Jekyl. This is the mechanism of trance-states of cerebral automatism, and even of absent-mindedness and intense attention. It may be applied to the explanation of an uncontrollable and consciously foolish fear or a hysterical paralysis.

We do not know the exact psychic or physiological process by which this blocking, and re-grouping of associations takes place. It is done by effort of the will, by fatiguing the attention, by the action of powerful emotion, by endogenous ferments perhaps, and by chemical poisons.

Whether the Freudian school accepts the dissociation theory I do not know, and it is difficult to find out. But if so it would be only a detail in their explanation of the mechanism of these same phenomena. With this school the forces of the subconscious would be the agent, which in struggling to fulfil desire caused blocking of the synapses, neural dissociations and the phenomena of altered personality, of hysteria and trance.

The Freudian school finds that the subconscious mind is the repository of a libido or desire whose urgings have been with varying success repressed and forgotten. Here are stored up the memories and instinctive trends of infancy, the emotional shocks and painful experiences of childhood. Though repressed and pushed back into the subconscious, yet they still exist as potent "complexes" and may be a source of troublesome mental states and bodily symptoms. The libido, unable to force itself into consciousness and express itself in the normal infantile way takes devious paths of activity and produces its obsessions, paralyses, etc., in trying to secure satisfaction. This is shown through psychoanalysis, by studying dreams and by various other diagnostic methods. The result leads to the view that the changed personality, the spasm, the anæsthesia, the paralysis, the exhaustion are the expressions of a suppressed wish.

This psychic mechanism offers a satisfactory explanation to some. It is a poetical, but not an adequate, practical or always safe method for the practising neurologist.

For to him as a clinician the causes leading to instability, obsession, the dominance of fear are not altogether or mainly subconscious. In most the influences of heredity, of a constitutionally weak brain, of physical disease, of obvious and conscious suffering, of maladjustments,

poor insight and defective education are the vitally important things, often not easily recognized but obviously to be treated. The subconscious psychic factor is certainly often only an extra and minor element. It is the final slight impact which makes an unstable structure fall. Often it is not subconscious really, and if present has only an academic and metaphysical importance.

Hence, I believe in treating the psycho-neuroses, by correcting the general instability, educating the inferiorly developed, removing irritations, and trying to make a perhaps rather poor human system carry its proper load.

True psycho-therapeutics is really a form of education or re-education. It belongs to no school, but is part of ancient medical art and physicians have known, and more or less applied, the method for centuries.

CONSTITUTIONAL INFERIORITY. PSYCHOPATHIC DIATHESIS

This is a term applied to a condition characterized by a congenitally unstable, nervous system, and more particularly an unstable mental condition. The patients, however, as a rule, are sufficiently under control of themselves and their morbid tendencies, to be responsible or partially responsible for their acts.

It includes persons who suffer from "nervousness," constitutional despondency and constitutional excitement and morbid instincts.

While constitutional inferiority may exist without developing any well-marked neurosis or psychosis, some episodical outbreaks often occur.

When the trouble is more especially associated with weakness of will, feebleness of mind, incapacity to decide or inhibit, or to initiate, it is in particular a *quantitative inferiority*. When the mind is not retarded or intellectually defective, but is dominated by morbid instincts (sexual or criminal) or unsocial trends, the person is more particularly a case of *qualitative inferiority* and he is then sometimes called a psychopathic personality. Various major psychoses may develop on this soil.

Constitutional inferiority exists as an independent state but it may underlie the following conditions:

Neurasthenia.

Psychasthenia.

Hysteria.

Hypchondria.

Dipsomania.

Compulsive and impulsive psychoses.

Persons with a constitutional inferiority may, under proper environment and educational guides, live fairly normal and useful lives. If,

however, they are brought up in such a way as to allow indulgence in abnormal impulses or habits; if they thus acquire vicious modes of living or go to excesses in any direction, they may develop criminality or some of the formal types of insanity, such as paranoia or even dementing types of insanity, like dementia præcox.

It is often somewhat difficult to say whether a person is simply a psychopathic personality, or whether he has not a constitutional inferiority in which criminal tendencies have been allowed to develop. It is often possible by the force of teaching and the influences of environment, to change the morbid tendencies, so that an inferior or psychopathic personality may become a fairly useful man. Great care should, therefore, be exercised in applying to a patient the term "constitutional inferiority" to excuse criminality, since there may have been only a slight tendency which has grown through indulgence.

HYSTERIA

Hysteria has been defined as the expression of a symbolic conversion of psychic into physical states. This condition in which the psychical is converted into physical is said by followers of the Freudian School to be an index of the aspirations of the race. For the manifestations of hysteria are thought to be the expression of subconscious instincts or desires.

Such a view need not interfere with a pragmatic method of dealing with the subject such as I adopt in the following description, in which the mental traits of the hysterical, and the physical phenomena they show are presented in clinical narrative without forcing into them philosophical and questionable mechanistic interpretations.

Hysteria is in its broadest sense not a specific disease but a morbid biological reaction, in which morbid bodily disturbances are caused by more or less subconscious mental states.

Hysteria is a widely prevalent quality of the human character. It displays itself in brief and often trivial episodes, such as unreasonable emotional explosions of crying and laughing, and in motor and visceral crises. These are often to be considered only as symptoms of a natural instability, and they are often associated with other real and organically morbid conditions. When this reaction is very severe, persistent and disorganizing, we have *major hysteria*, which is distinctly a disease or fixed symptom-group having a definite origin, course and symptoms.

A slight degree of this hysterical reaction is very common and can hardly be called abnormal. Nearly all women are said to be somewhat hysterical.

When the condition occurs occasionally and under stress of emotion, it is episodical or *minor hysteria*.

Etiology.—Of the predisposing causes of hysteria in general, heredity is the most important. In about 75 per cent. there is a history of hysteria, or some neurosis or psychosis in the parents. The disease is transmitted more often by the mother. Heredity is particularly apt to be important in the hysteria of children; it is a much smaller factor in hysteria of adult males. Hysteria is a disease of early adult life, most cases occurring between the ages of from fifteen to twenty-five in females; it occurs later in males. Hysteria attacks children between the ages of eight and fifteen, chiefly between eleven and fourteen. The trouble affects women more than men in the proportion of four to one, varying much with race, climate and occupation. Hysteria major, however, occurs oftener in men than women; it occurs in adult and middle life and there is often no hereditary history. These statements are based on personal experience of this type of hysteria as seen in this country. They do not correspond with the observations of European observers or of alienists, but are certainly true for the type of disease which I am describing. Hysteria occurs in all classes of life, but rather less frequently in the middle classes than among the poor and the very rich. Male hysteria is more frequent in the poorer classes who are subjected to the influences of alcoholism, poverty, injuries, etc. Hysteria is certainly much less frequent in its severer forms in this country than in some parts of Europe, particularly France. In my experience it is much less frequent than epilepsy in the northern and eastern parts of this country. It occurs, however, quite frequently in the negroes and also in the Latin races of this country. Bad methods of education and bad sexual habits undoubtedly tend to promote the development of the disease.

The most important single exciting factor is powerful emotion, particularly fear. Other emotions of an allied character—excitement, sorrow, anxiety—may bring on attacks. The disease can be developed by imitation. Injuries combined usually with mental shock are fruitful causes of producing hysteria. The infectious fevers, syphilis, diffuse hemorrhages, the poisons—lead, mercury, and tobacco—the administration of ether, mental and bodily and sexual excesses, are all important agents in developing the disease. Acute alcoholism is not very rarely followed by hysteria.

General Symptoms.—*Mental.*—Mental hysteria shows itself clinically in certain morbid moral manifestations. The typical moral hysteric, for example, is intensely self-centred, craves for sympathy and is desirous of being always in the centre of the stage. To secure her ends she exaggerates symptoms, tells lies, practises cunning deceits and constructs false and ingenious stories. Such patients, in order to gain their end and excite human sympathy, will even injure and mutilate

themselves or will prevent the healing of their wounds. In fact, all manner of devices are used to make it appear that they have some ailment or painful condition which demands continual attention. Without her headache, or exhaustion, or neuralgia, the hysterical woman knows, or her subconsciousness knows, she would not count in the household for more than she is worth there.

Aside from the peculiarities of the hysterical mind along the moral lines, the hysterical person develops other characteristic mental phenomena. These consist of dreamy states, periods of amnesia and automatism and also disorders of mentality which go by the name of hysterical insanity.

The great proportion of patients who have disturbances of bodily function such as paralyses, anæsthesias, neuralgias, may be practically free from the exaggerated form of moral hysteria, and appear only perhaps rather dull and depressed, nervous and apprehensive.

Physical.--The various forms in which the somatic phases of hysteria show themselves may be grouped as follows:

Convulsions; tremors; clonic and tonic spasms, paralyses of the type of monoplegia, hemiplegia and paraplegia; neuralgias and anæsthesias.

There are also vasomotor and secretory disturbances; and disturbances of the autonomic system shown in vago-tonic and sympathetico-tonic attacks.

The reflexes are to some extent disturbed.

Special Types.—Hysteria minor or the hysterical temperament is characterized by an interparoxysmal condition of emotional weakness, nervousness, hyperæsthesia and pains, and by crises of an emotional, motor, sensory or visceral type. In hysteria minor there are no permanent objective marks like anæsthesia and paralysis, and rarely convulsive seizures. The patient, who is almost always a girl or young woman, gradually develops an undue sensitiveness, the mind is depressed, and she gets easily alarmed. She has feelings of nervousness and lacks control over the emotions, she laughs and cries very easily and yields to every impulse. She suffers from headaches, which are usually diffuse and often severe and chronic, and from spinal pains. She sleeps, as a rule, rather badly and often has disagreeable dreams. She has, under any little excitement, sensations of tickling, fullness or choking in the throat, forming the condition known as *globus*. Excitement also brings on attacks of trembling or chilly feelings which come and go. There is more rarely a considerable amount of vasomotor instability, as shown by flushings and by coldness of the extremities, and vago-tonic attacks.

She has with more or less frequency distinct crises of an emotional character, during which she laughs or cries without apparent cause, or at least to an extent beyond her control. She may have attacks of

vomiting or headache, or of intense mental excitement amounting almost to delirium. In some cases the patient has somnambulic attacks at night, or she may have under a little excitement attacks of cerebral automatism during which she involuntarily does things that she is entirely unconscious of when she comes out of the attack. The crises are followed by a copious discharge of very light urine.

With all these symptoms there may be the moral traits above described.

Hysteria major is characterized by interparoxysmal manifestations of anæsthesia, paralyses, contractures, tremors, peculiar mental conditions, and by paroxysms of an emotional, convulsive, or other serious nature. Hysteria major is what is usually meant when one speaks of hysteria as a disease; it includes also hystero-epilepsy. The onset may be gradual, but not infrequently it follows some shock, the first symptom being a convulsion, a paralysis, or some emotional outburst.

The crises of hysteria may be: Psychical; motorial; sensory; visceral; angio-neurotic.

The most common of the paroxysms of hysteria are emotional outbursts of crying or laughing; after this come motor disturbances in the shape of convulsions of various types or of hemiplegia or other form of paralysis. Besides this we have attacks of severe pain, forming neuralgic crises; attacks of nausea, gastralgia and vomiting, forming gastric crises; much more rarely there are prolonged attacks of hysterical coughing, hiccoughing, sneezing, or rapid breathing. The hysterical seizure may also take the form of attacks of trance and lethargy, catalepsy amnesia, and cerebral automatism.

The emotional crises are episodes of the same nature as occur in minor hysteria.

Hysterical convulsions have two rather well-defined types. One of them is the ordinary form of hysterical convulsions; the other is a much more severe disturbance in every way and is known as a hystero-epileptic or hysteroid attack. In the hysterical convulsion the patient, under the influence of some excitement, injury, or acute gastric disturbance, rather suddenly falls down and begins to go through various irregular movements of the body, such as thrashing with the arms, kicking with the legs, throwing the head from side to side, rolling about on the bed or floor. In the more distinctly convulsive seizure the hands and arms and fingers are flexed, the legs and feet are extended, the eyes are generally closed, the eye-balls often converged or moved about irregularly, the pupils dilated. There is some lessening of sensation over the body and of the conjunctivæ. The patient often utters noises or screams at intervals. She may bite her lips, but does not bite the tongue, nor does she ever hurt herself in her various contortions. The attack may last for half an hour to several hours, unless some measures are taken

to break it up. In other forms of hysterical convulsion there is simply a general shaking or trepidation of the body as though the patient had a chill; in other cases again the main type of movement is that of opisthotonos, the patient rising up upon the head and heels and arching the body in as tetanus. Again the attack may consist simply of a little rigidity of the body or of a series of rhythmical movements of the head or trunk or limbs, the patient sitting up and oscillating the head or swaying the trunk or moving the arms, uttering at the same time incoherent words. In still other cases the patient simply falls down and lies unconscious, like a person sleeping, for a few minutes or even an hour. In children the attacks may be associated with peculiar noises and movements in imitation of animals, such as the growling of a dog or the mewing of a cat. This condition is called therio-mimicry. In some instances the attack may be accompanied by or may end in a condition of mental excitement approaching delirium. The patients while suffering from these seizures generally appreciate what is going on about them, and will often respond to some stern order for them to cease or will be brought to a state of quietude by pressure upon some part of the body which provokes pain. In women in particular, pressure over the ovaries or epigastrium will abort the attack; the application of cold water or an emetic will do the same.

After a hysterical crisis or sudden shock, the patient may be found to have a paralysis of arms or legs or one side of the body.

The Symptoms of the Interparoxysmal State.—Between the crises of major hysteria the patient may be in a fair condition of general health, but usually presents certain definite chronic manifestations of the disease.

Sensory Symptoms—These consist of superficial and deep hyperæsthesia and anæsthesia and anæsthetic disturbances of the special senses. When all these forms of anæsthesia affect one side of the body we have a typical major hysterical condition which may last for years. Cutaneous anæsthesia occurs in three forms: the common form is that of hemianæsthesia involving one-half of the body; next in frequency is the segmental anæsthesia involving an arm or a leg or part of the face or head; rarest of all the forms is a disseminated anæsthesia occurring in the form of patches. These various modes of distribution are shown in the accompanying figures. The anæsthesia is a pain anæsthesia chiefly. The tactile and thermic sensations are less markedly affected. Loss of deep sensibility is rare. The anæsthesia is in some cases transferable by means of magnets or electrical irritants or by suggestion. The anæsthesia can also be lessened or removed temporarily by the application of magnets or coins or pieces of metal. For example, if a silver coin is fastened upon the anæsthetic area, in the course of a few minutes

or a few hours there will be a zone of normal sensation under and around the coin. Sometimes the temperature of the skin upon the anæsthesic part is lowered 3° or 4° F., and upon pricking the skin blood does not flow. The anæsthesia is oftener upon the left side in the proportion of three to one. Hysterical anæsthesias are not accompanied by subjective sensations as are organic anæsthesias. The skin reflex is usually abolished, as is the conjunctival reflex. The deep reflexes are lessened or unchanged. Anæsthesias of some kind occur in a very large proportion of the serious forms of hysteria major. They are rare, however, in children and are rarer in women than in men, in the author's experience. Anæsthesia of the mucous membranes is present chiefly in hemianæsthesia; it then involves the mucous membrane of

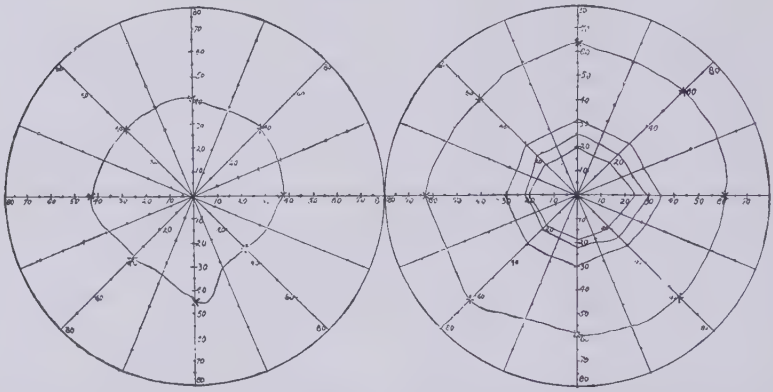


Fig. 226.—Hysterical loss of color sense and limitation of visual field. Color sense absent in left eye and field contracted; in right eye field less contracted; order of appreciation of colors from without in was yellow, violet, blue, red, green.

the mouth and throat, and to a less extent that of the nose and glottis. Hemianæsthesia is usually accompanied by some hemiplegia and often by some tremor. Segmental anæsthesia is also often accompanied by some degree of paralysis of the part.

Visual anæsthesias. One of the most common of the permanent stigmata of hysteria is a concentric limitation of the visual field and a disturbance in the color sense. Complete loss of this sense may take place or there may be a variation in the ways in which the colors are perceived. There may be also a distinct diminution in the acuity of vision or even a complete loss of sight of one eye or both eyes. This hysterical amblyopia usually comes on suddenly and may last for weeks. It is not always a true blindness and the patient left to walk about a room does not run into obstructions. The visual disturbance is most common

with hemianæsthesia. It is more marked on the affected side, but exists to some extent on the healthy side. The limitation of the fields is shown in the accompanying cut (Fig. 226).

Hearing. There is sometimes a diminution in the acuity of hearing of one ear, and this occurs, if present, in connection with hemianæsthesia. There may also be a loss of hearing to high and low notes, while hearing is apparently fairly good to notes of medium range; and finally there may be a diminution in hearing by bone conduction, while hearing by aerial conduction is but little impaired, this being due probably to an anæsthesia of the acoustic nerve.

Disturbances of taste in the form of anæsthesia or paræsthesia are quite frequent and are important signs in hysteria. The loss of the sense of taste may involve only the back portion of the tongue and the palate.

The sense of smell may be abolished, but this usually occurs in connection with hemianæsthesia.

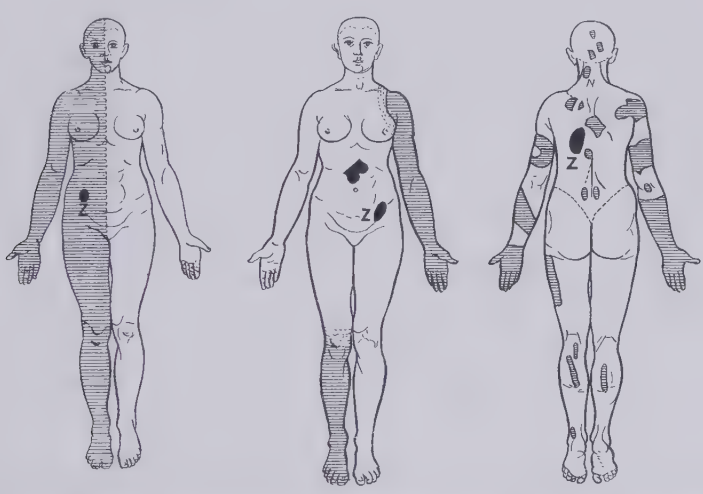


FIG. 227.—The three types of distribution of anæsthesia in hysteria; hemianæsthesia, segmental and disseminated. *Z*, hysterogenic zones.

Hyperæsthesias and neuralgias. Pure neuralgias are somewhat rare in true hysteria, but hyperæsthesia and pains of various kinds are not at all infrequent. Hyperæsthesia occurs in the form of patches at different parts of the body. These sensitive points may, when pressed upon, bring on paroxysms of various kinds, and they are therefore called the hysterogenic zones (Fig. 227). The most common seat of these zones in women is over the ovaries; in men, in regions corresponding to the ovaries and on the scrotum. Hysterogenic zones, however, may be found

just beneath the mammary gland, on the epigastrium, along the spine, and in other places. These zones are sensitive areas; they can be made to disappear by applications of electricity and by refrigeration and counter-irritation. Hysterical patients often suffer from local headaches, which are apt to be confined to the top of the head or to the sides near the temples. The pain is severe, sharp, and boring, and may exacerbate with such intensity as to produce symptoms almost resembling meningitis. The spot-like pains are known as hysterical clavus. There may be also a peculiar persistent local pain of a similar nature to *clavus* called *topalgia* or place-pain. Hysterical patients occasionally have migraine, facial neuralgia, and intercostal neuralgia.

Much more often they have pains along the spine, producing symptoms of spinal irritation. Hysterical patients also have at times attacks of vagatonia and of palpitation and pains over the heart, constituting what is known as pseudo-angina. Such troubles are much more frequent in women.

Arthralgias are painful joints in which the condition simulates a chronic arthritis but has none of the evidences of inflammation, except sometimes a slight œdema or effusion.

Motor Symptoms.—The motor symptoms of hysteria are paralyses, myasthenia, contractures, tremor, and choreic and ataxic movements. The paralyses of hysteria take the form of hemiplegia, paraplegia, and monoplegias. Hysterical hemiplegia occurs usually rather suddenly, often as the result of some severe shock. The left side is more frequently attacked. The arm is most affected, the leg next, while the face is hardly ever involved. The paralysis is not an absolute one, and the patient is able to drag himself along. The deep reflexes are usually not exaggerated and they may be for a short time absent. The paralysis is thus a flaccid one. The gait of the patient is different from that of hemiplegia due to organic disease; in hysterical hemiplegia the patient drags the paralyzed leg after him, in organic hemiplegia the patient swings the paralyzed leg around in a half-circle (Fig. 228). This peculiarity of the gait, the absence of abnormal reflexes, the absence of paralysis of the face, and the presence very commonly of other hysterical stigmata are sufficient to enable one to make the diagnosis. Sometimes the face on the affected side is slightly drawn by a spasm, so that it appears to be paralyzed when it really is not (Charcot). Monoplegias affect the arm or leg, very rarely indeed the face, occasionally the eye muscles, and most commonly of all the muscles of the larynx. Hysterical monoplegia is usually accompanied by anæsthesia of the affected part and by other symptoms of hysteria.

In children hysterical palsies are almost always monoplegic, never

hemiplegic. There are no serious atrophic changes or disturbances of the electrical reactions. Hysterical eye palsies show themselves in the form oftenest of an insufficiency of the internal recti. In hysterical palsy of the larynx the adductors are psychically involved so that the patient cannot speak loud, and the condition is called *hysterical aphonia*. The trouble often comes on suddenly, the patient finding that he cannot speak above a whisper. The paralysis is mental and therefore the adductors can be approximated in coughing. The trouble is distinguished from

FIG. 228.—Hysterical hemiplegia showing flaccidity of the paralysis.—(*Loude.*)

laryngeal inflammation by inspection of the affected part. The tongue and other muscles of articulation are in very rare cases also involved, and hysteria may produce symptoms resembling a bulbar paralysis. Paraplegia is a rather uncommon form of hysterical palsy; it is usually brought on by emotions of depressing character, often associated with some slight injury. It may be accompanied by a good deal of pain in the back, and the form of disease which was once known as "spinal concussion" consisted in many cases of hysterical paraplegia combined with traumatic disturbance of the spine. In hysterical paraplegia there

is very little wasting of the limbs and no change in the electrical reactions. The deep reflexes may be somewhat increased or normal; they are never absent. There is never any prolonged or persistent ankle clonus, but there may be a short or spurious clonus due to a general exaggerated irritability of the nervous system. The sphincters are never involved except temporarily or through some complication.

Myasthenia is a frequent, peculiar, and interesting symptom occurring in the interparoxysmal stage of hysteria. It consists of a more or less temporary feeling of weakness of an arm or of the legs or whole body.

FIG. 229.—Hysterical contracture of hand.

Thus a person in lifting a dish from the table suddenly feels the arm give out, and if not careful the dish is dropped; or while walking the patients suddenly feel as though they had lost all power in the lower limbs. This myasthenic condition is generally temporary, but it may be so permanent as to produce a certain degree of monoplegia or paraplegia. The myasthenic condition may precede a paralysis. It presents no objective signs in the way of electrical reaction; it involves a whole member, not a single group of muscles.

Contractures.—In some forms of hysteria there is a tendency for the muscles to undergo contracture under slight mechanical stimulation, much as pressure or a blow. This tendency to contracture in hysteria is called the *contractural diathesis*, and it is an important sign. The contractures may be temporary, disappearing soon after the exciting cause ceases, or they may develop independently and last for a long time. They usually but not always disappear in sleep, and under anæsthesia. They involve the legs, arms, and facial muscles, and may

be associated with paralysis and anæsthesia (Figs. 229, 230). The contracture is usually a spasmodic, not a paralytic one.

Tremor occurs in hysteria in a considerable proportion of cases, more especially those in which there are hemiplegia and hemianæsthesia. Hysterical tremor simulates all the various types. The common form

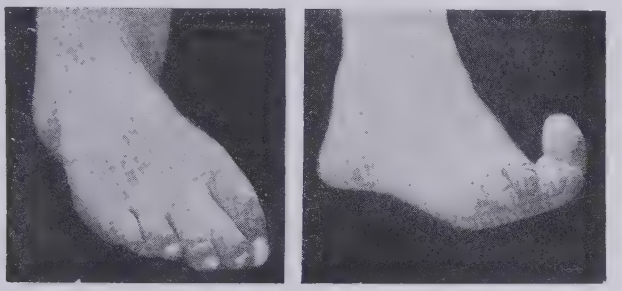
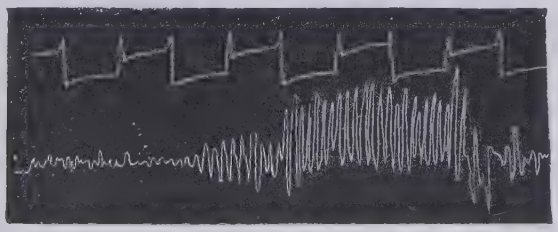


Fig. 230.—Hysterical contracture of toe.

is one in which the oscillations occur from five and a half to seven and a half times a second, and is jerky and irregular in type. It ceases for a time when the person is quiet or lies in the horizontal position; also during sleep. It affects the head and tongue as well as the extremities, the latter more upon one side than the other. It may be chiefly in the lower limbs. Sometimes it has the type of an intentional tremor, ceasing on rest of the hand and increasing when the hand is moved, as



Repose. Movement.

Fig. 231.—Illustrating intentional tremor in hysteria. (*Tourette.*)

in raising a glass to the lips (Fig. 231). It then resembles exactly the tremor of multiple sclerosis. There may be a slow tremor of four to five and a half oscillations per second; this persists during rest and is but little modified by voluntary movements. It imitates the tremor of paralysis agitans. Finally, there may be a rapid tremor of eight to nine oscillations per second. This also persists during repose and is but little modified by movements. It imitates the tremor of Basedow's disease, alcoholism, and neurasthenia.

Trophic Disorders.—In hysterical paralysis a slight amount of atrophy occurs, but only such as would naturally follow disuse of the part. Cutaneous eruptions and dystrophies practically do not exist or, if present, are the result of complicating disorders.

Visceral Symptoms.—Hysterical patients often suffer from dyspepsia and constipation, also from anorexia, and in some cases from vomiting or regurgitation of food. Occasionally the anorexia and vomiting become persistent; the patient refuses food or rejects all that is taken; she emaciates, becomes weak and bedridden, and develops into that particular phase of hysteria known as *anorexia nervosa.* In these cases, along with the aversion to food and vomiting, there may be a great deal of gastralgia.

The urine in hysteria is apt to be of low specific gravity. Sometimes there is retention of urine; in extremely rare cases there is a condition known as ischuria and anuria, in which for several days extremely small quantities of urine are passed, owing apparently to a suspension of the functions of the kidney. Such cases should always be carefully investigated.

Vasomotor symptoms are very common. The autonomic and sympathetic tonus is often involved, causing vaso-vagal attacks and gastro-intestinal disturbance with pain, diarrhœa and vomiting. These disturbances of the sympathetic are also shown by flushings and pallor, cold extremities, and at times an œdematous condition of one or more extremities. This œdema may be of the ordinary pale, waxy character, pitting upon pressure. In other cases it has a peculiar bluish tinge and it does not pit; the hands, which are the parts generally affected, are several degrees below the normal in temperature, and the limb resembles in some respects the condition in Raynaud's disease. Gangrene, however, never supervenes. This form of œdema is known as the *blue œdema of hysteria.*

There occur in hysteria febrile attacks, and much has been written upon the subject of *hysterical fever,* which does not exist.

Hystero-epilepsy.—The form of hysteria which shows itself by the development of severe crises known as hystero-epileptic attacks is extremely rare in this country, at least in its typical phase. It has been particularly studied by the French writers Charcot, Richer, and others. Hystero-epilepsy, as this form of the disease is called, is a true hysteria and not epilepsy at all, nor a mixture of hysteria and epilepsy, though the name would suggest that that was the case. The typical attacks of hystero-epilepsy begin with certain prodromata consisting of a feeling of malaise and irritability which may last for several hours or a day.

There is next the epileptoid phase, lasting from one to three minutes;

third, a phase of contortions and grand movements, lasting one to three minutes; fourth, an emotional phase, lasting from five to fifteen minutes; and, finally, a stage of delirium, lasting a variable time; the whole attack lasting from five to twenty minutes. In this country we occasionally see hysterical patients exhibiting one or two of these phases, but very rarely indeed do they ever go through the whole series. The patients who suffer from hystero-epileptic attacks generally during the interparoxysmal stage present many of the stigmata of hysteria, such as paralyses, contractures, and anæsthesias.

Hysterical crises which take the form of convulsions or emotional seizures sometimes end or are associated with attacks of *catalepsy* or *trance* or attacks of amnesia and cerebral automatism. As these conditions all occur in other diseases than hysteria, they will be described elsewhere in connection with the subject of the disorders of sleep and of consciousness.

Hysterical persons occasionally are attacked with violent and persistent hiccoughing or sneezing. Sometimes also there come on attacks of extremely rapid breathing or hysterical polypnœa, during which the respirations run up to fifty or seventy a minute. A hysterical cough sometimes occurs; and lasts for a long time. Œsophageal spasm with consequent dysphagia is another one of the somewhat rare phenomena of hysteria. Indeed, many of the symptoms referred to in this last paragraph may be grouped as tics or *morbid mental habits*, and not truly hysterical.

Pathology.—There is no known anatomical change at the basis of hysteria. We do not find the marks of degeneration as we do in serious forms of insanity and epilepsy.

Diagnosis.—The essential characteristics are the peculiar emotional condition of the patient, the past history of hysterical crises, the presence of the stigmata of hysteria, such as anæsthesias, limitation of the visual field, paralyses, and contractures. The variability of the symptoms, their susceptibility to influence under suggestion and rigorous moral measures, the absence of organic disease or of serious disturbance of nutrition, the sex and age, and the cause should also have weight in guiding us to our decision.

The test of hysteria minor is simply the presence of a morbidly easy reaction of the mind upon the body. It has no absolute criteria. What is morbid must be determined by the judgment of the physician considering the education and environment. *Hysteria major* is a rare disease and has a clear-cut symptomatology. It has crises and the stigmata already described.

Hysteria and Paychasthenia.—If a person has an obsession or fixed idea which consciously affects his mind and actions, but not directly his

body or functions, he has psychasthenia, or an obsessive psychoneurosis. The obsession in hysteria is usually a subconscious one; in psychasthenia it is conscious.

Diagnosis of special symptoms of hysterical manifestations. Hysterical paralysis is characterized by the fact that there is no marked degree of wasting of the muscles, no electrical reactions of degeneration, the deep reflexes are preserved or exaggerated, and other marks of hysteria are present. Hysterical anæsthesia can generally be lessened over certain areas by the application of the magnet or can be made temporarily to disappear; it is peculiarly distributed in the way described under symptoms and is associated with anæsthesias of the special senses. Hysterical contractures sometimes cease during sleep and generally under deep narcosis, and the use of an anæsthetic may clear up the case. They usually follow a fit, an injury, or an operation. They are somewhat increased on attempts to overcome them by force; they are usually associated with paralysis and anæsthesia and other hysterical symptoms.

Hysterical convulsions. These differ from convulsions of epilepsy in the way best indicated by the following table:

Hysterical Convulsions	*Epileptic Convulsions*
Brought on by the emotion or injury; rarely any aura; no initial cry; movements co-ordinate; tongue not bitten, and patient never injures herself. Duration perhaps several hours with intermissions; consciousness generally preserved. Micturition and defecation do not occur. No rise of temperature; may be stopped artificially. Rarely occur in sleep.	The opposite in all these particulars.

Although the differentiation here made usually holds good, there occur psychogenic convulsions in which all the phenomena of the epileptic fit are present.

The hystero-epileptic attacks are so characteristic that a mistake could not be made.

Prognosis.—The prognosis of hysteria in children is good. They generally get well, though in some cases there is a recurrence later in life. Minor hysteria under proper treatment usually gets well or at least is of little importance. The major forms when chronic are often intractable even under the best treatment. When a severe form of hysteria occurs in a person of feeble frame who is surrounded with a sympathetic family, the task of rescuing her from her disorder is a very arduous one. Traumatic forms of hysteria which are not infrequently associated with some actual physical injury are sometimes difficult to cure. Hysteria which is associated with some organic disease, such as

a severe pelvic disorder or an organic affection of the central nervous system, has a poor prognosis. Hysteria in the male is generally curable, but it requires vigorous treatment, and spontaneous cure is by no means likely to happen. Major hysteria has a vastly better prognosis if proper treatment for it is instituted early.

Treatment.—The treatment of hysteria may be divided into mental, mechanical dietetic, and medicinal measures.

By all odds, the most important factor in the treatment of hysteria is the mental treatment, and the most important primary measure to be taken is the isolation of the patient. She should be placed where she will not be surrounded by sympathetic friends; where life will be a regular one; where some occupation may be given which will engross attention, interest the mind, and call into play the physical activities.

In the major forms of hysteria associated with anorexia, emaciation, anæmia, and possibly pelvic disorders, the "rest cure" as elaborated and carried out by Weir Mitchell forms by all odds the most successful means of treatment. In many cases of less severe character a partial rest cure in which the patient is separated from her family but is not placed under such severe restrictions may be all that is needed. In the case of children removal from home is often advisable, and the discipline of well-conducted schools is a most excellent measure. Re-educational treatment by therapeutic talks, by direct and indirect suggestion, by occupation, work, marriage, learning a craft or business; life in a sane environment, these are the real methods of cure. Psychoanalysis and the method of Freud have their adherents.

The mechanical means used in hysteria are hydrotherapy, electricity, massage, and exercise, and they have symptomatic value. Of these measures hydrotherapy and electricity take the first rank. In hydrotherapy the douche or jet to the back, the shower and cold plunge, and the half-bath are the most efficacious. In the electrical treatment the static and faradic currents give some results. The static sparks often relieve contractures and lessen or remove the anæsthesias, and both forms of electricity seem to have a generally beneficial tonic effect. Doubtless the psychic element is the most important factor in this as in other mechanical measures. Massage is of some value in promoting nutrition and it also has a favorable sedative effect on many cases. Exercise, particularly of an active kind, such as stimulates the mind and interests one, is a measure of extreme value and one which has perhaps not been sufficiently appreciated.

The drugs which can be recommended in hysteria are not numerous and their power is limited. Valerianate of zinc, asafœtida, iron, and the bromides are the most important of the nervines. In hysterical children a capsule containing two grains of valerianate of zinc and one of sulphate

of quinine is often efficacious. Gowers places more reliance upon the oil of turpentine in doses which should be increased to the point of strangury.

In the treatment of hysterical convulsions the most efficient measure is the administration of an emetic, and this can be best done by giving hypodermically one-twelfth or more of a grain of apomorphine. Convulsions can be stopped sometimes by throwing water in the face or on the epigastrium; by firm and somewhat long-continued pressure over the ovaries; by the administration of valerian, aromatic spirits of ammonia, or compound spirits of ether.

CHAPTER XXII

PSYCHASTHENIA

Psychasthenia is a chronic, non-dementing psychosis, characterized by the occurrence of imperative ideas by compulsive and impulsive acts, by morbid fears and conditions of agitating doubt. The term obsessive neurasthenia or obsessive psycho-neurosis is used by some instead of psychasthenia. Beard originally described many of these symptoms and classed them under the head of neurasthenia. Indeed, neurasthenia usually accompanies the condition, and many unstable people, when they get weakened or exhausted by work or disease, develop morbid fears and obsessions. So that part of the cure of psychasthenia is to treat the neurasthenia.

Etiology.—The most frequent etiological factor is heredity; nearly one-half of my cases gave a history of direct or indirect mental disorder in the ancestry. I have seen several cases in which the obsessive psychosis occurred in mother and son or mother and daughter.

Lues, tuberculosis, and alcoholism occur in the ancestry, but not so prominently as in other psychoses. The disease occurs in males and females about equally. Symptoms of the trouble may be shown in children as young as eight or nine years; more than half the cases, however, develop between the ages of fifteen and twenty-five; nearly one-fourth of the cases between the ages of twenty-five and thirty, while one-eighth of the cases develop between the ages of ten and fifteen. A few cases occurred between the ages of thirty-five and forty, and it is seen again with some frequency at the time of the climacteric. I have seen three cases in which the disease developed after the age of sixty. The earlier the case develops, the more persistent, as a rule, are the symptoms liable to become.

Among the most frequent exciting causes may be mentioned some kind of shock by fall or accident; next after this is alcoholic excess. Among other causes I have noted infectious fevers, an attack of chorea, a stroke of lightning, car sickness, educational strain. Engagements, marriage and pregnancy have been rather often a starting point of the disease.

Symptoms.—The symptoms develop in four different types, though these are often intermingled, and, in most cases at least, three of the

characteristic groups of symptoms are more or less present.[1] These types are those characterized by (1) morbid fears, (2) by imperative ideas, (3) by a doubting mania, and (4) by morbid impulses.

In 91 cases I find the order about as follows: Morbid fears, 37; obsessive and fixed ideas, 25; doubting manias, 18; compulsions and impulsions, 11. Psycho-analysis may show that these different groups have a different mechanism, but my experience is that they are clinically often allied and intermingled.

Morbid Fear, Doubting Mania.—This disorder begins usually rather suddenly as a result of some slight shock, and perhaps of some causal and unimportant incident. A young man, while in church, or in a crowd, suddenly has a feeling of disturbance of consciousness, with something akin to vertigo. He is alarmed, the head seems full, the heart palpitates, and he feels as if he would faint. These attacks may be repeated. They have been called psychic seizures, or psycholepsy. Some of these seizures appear to be psychogenous vago-tonic attacks. After one occurs the patient becomes afraid to go in a crowd (agoraphobia) or in an enclosed place where there are people (claustrophobia; he may even be afraid to go out of doors without a companion. This state is accompanied with some general nervousness, depression, insomnia, and pressure or pain at the back of the head or vertex. He is often unable to pursue his work or study, and becomes worrying and introspective. After some months or a year the secondary symptoms disappear, but he may for several years feel a dread of going in certain places.

Fear of storms and of lightning (astraphobia) may come on in much the same way, but this form of morbid fear is less disturbing as a rule, and often the patients are quite well as long as the sky is reasonably clear. Some patients are so sensitive, however, that they become nervous and depressed before storms and can predict them as unerringly as do rheumatic or neuralgic sufferers.

In fear of contamination and dirt (mysophobia) the patient is constantly washing the hands, changing the clothes, and trying to avoid contact with people. The sufferer will spend an hour or more in dressing and undressing or in preparing to go out. After a time almost all activity is curtailed by dread of contact with something, and the victim sits in a chair, or lies in bed with hands enveloped in cloths to protect him from the dust. All the interests of life are lost in this one absorbing idea and fear, though the patient is not demented or maniacal.

[1] The condition of obsessive and fixed ideas has been called *compulsive insanity*. Compulsive acts may result, but they are accompanied by consciousness of the nature of the act, by feelings of anxiety, and by attempts at resistance. The victim of morbid impulses is said to be suffering from *impulsive insanity*. Here the impulses spring suddenly into consciousness, and acts follow at once without the patient being able to resist them. These impulses vary in character and severity from the impulse to touch a lamp post to that to commit a murder.

There is a decided depression, but not a true melancholia. If the patient, however, is driven by his attendants to do things he dreads, he cries and becomes excited and hysterical.

Fear that the heart may stop or the breathing cease are also serious types of morbid fear. The patient is always feeling the pulse and asking for an examination of the heart and an assurance of its soundness.

The number of fears is almost as great as the number of objective things. The more common types are mysophobia, agoraphobia, claustrophobia, fear of riding in cars or carriages (in New York City of subways) and astraphobia. There are also many "occupation fears," such as that in which the barber fears to use his razor, the tailor to cut the cloth, the bartender to mix drinks, and the business man to sign his name to checks. These symptoms run a course of one-half to three years. If properly treated, the patient recovers, with a tendency to relapse.

Allied to the morbid fears are the morbid "philias" or anxious concern for the safety and health of the race, more particularly of the domestic animals. The patients cannot walk the streets without concern over the tail of the docked horse and will get up at night to fetch in a wailing cat which seems to him in real, not amorous, distress (zoophilia).

While morbid fears occur in very early life, often developing at puberty, doubting mania occurs rather later, usually after adolescence. The doubting habit is often a morbid outgrowth of an original morbid scrupulosity and attention to detail. The patient has usually been very orderly in habit and worried when things are not arranged or done in a very orderly way. The clothes and bureau drawers and the bric-a-brac of the room have all their set place, and a discomfort is felt at seeing them out of the regular spot. Then some disturbing emotion or sickness sets agoing an absolute distress of mind if things are not just so or are not perfectly clean. Then begin frequent washings, change of clothes, an hour is spent in arranging the articles on a bureau and mantle or in dressing the hair.

The doubting mania sometimes seizes girls—more rarely men—who are about to be married, and engagements are repeatedly made and broken. Often the doubting habit is shown in connection with fear of fire, of burglars, or gas. The patient gets up at night a dozen times to feel the gas jets, to see if the doors are locked, to look under the bed, and do other whimsical things. These doubting states are often not very serious, and are only exaggerations of a naturally timid and scrupulous disposition. Alcoholism is at times associated with doubting mania.

Obsession, Fixed Idea.—Obsessions and fixed ideas often relate to some rather trivial thing. In the obsessive constitution the association

machinery seems to get clogged and ideas tend to stick pertinaciously in the mind. A slight suggestion or remark casually made with some especial rhythm or emphasis stays and bothers the patient. More often there are one or more ideas which stay and torment him. They are unpleasant ideas of possible mistakes made or injuries thought to have been done. They are usually absurd, and the patient knows perfectly well that they are so, but they cannot be shaken off. They dominate and harass the sufferer who feels really like one "possessed." They lead to insomnia, nervous restlessness, hysterical outbreaks, and crises of the abdominal viscera, with diarrhœa and polyuria. Yet often the patient, if naturally intelligent and sensible, will keep himself in control, and show most of the time no emotion or evidence of suffering. These fixed ideas may attack a person periodically for years, or may after a few months disappear, not recurring at least to any extent. Shocks, acute sickness, and depressing experiences will bring them out.

The syndrome of the fixed idea, not reaching the intensity of a delusion, may be part of a melancholia of involution or of manic-depressive insanity.

Morbid Impulse.—Impulsive psycho-neurosis is the condition in which impulses spring suddenly into consciousness, and acts follow at once without the patient being able to resist them. The morbid impulses may be very slight and harmless, such as a desire to touch every lamppost passed on the street, to step on alernate stones of the pavement, to put the left foot first in going from a room, or they may lead to violent acts, such as murder, theft, or arson. When there is a morbid impulse to utter certain words over and over, it is called onomatomania. Psychic tics belong to the morbid impulses. A patient of mine, a clergyman, who was overworked and anxious, developed impulses to an automatic coprolalia and profanity while at work in his study, and even while preaching. Another clergyman had the same tendency along with a decided doubting mania.

Morbid Compulsion.—When a morbid impulse rises into consciousness and is held more or less under restraint it is called rather artificially a *compulsion.* A patient while shaving started to cut himself. He never did do this, though he was obliged to give up doing this function himself for a time. He also felt an intense desire to jump overboard while on a ship, though he had had no melancholia at any time. The compulsion is often perfectly restrained, and I have known a man who went about for years with at times an almost overwhelming fear that there would come upon him an uncontrollable impulse to assault some one.

Compulsive and Impulsive Manias.—When impulses and compulses involve a larger psychical sphere, we have the various conditions known as kleptomania and pyromania, homicidal and suicidal mania. But

here, along with defective inhibition, there is often a morbid degree of criminal instinct or there may be present another psychosis, such as mania or melancholia or paranoia. Thus the sexual criminal acts and perversions are of the class of compulsions or impulsions, plus a congenital and hereditary instinctive defect.

A type of impulsion midway between the mild and neurasthenic and the deep-seated psychopathic cases are the impulses to drink leading to periodical inebriety. Here there occurs periodically, with or without the excitement of a single taste of liquor, an overwhelming impulse to drink, and the patient drinks until consciousness is lost.

In fine, impulsive and compulsive insanity are generally part of the psycho-neurosis but they may occur as episodes in melancholia, mania, dementia præcox, paranoia, and the criminal constitution.

Hypochondriasis and Psychasthenia.—Hypochondriasis is a term applied to a morbid mental condition in which the patient thinks he is suffering from some physical disease. It is a much used and much abused term. Hypochondriacs vary much in the degree in which the symptoms show themselves. In some cases the belief in the disease is so foolish and extravagant as to form a real delusion, as when a patient thinks he has a worm in the head or stomach or an absolute and permanent stoppage of the bowels. In these cases the hypochondria is only a manifestation of a melancholia, generally a chronic melancholia of involution.

In more cases the patient's hypochondria is simply a manifestation of a morbid fear or an *idée fixe* or obsession. These are cases of psychasthenia. A mild degree of hypochondria often characterizes chronic neurasthenia. Hypochondriasis, therefore, is only a syndrome which occurs in melancholia, psychasthenia and neurasthenia, and is not a separate disease.

Hysteria and Psychasthenia.—Some at least of the mental traits and emotional crises of hysteria are forms of or episodes of psychasthenia. There is here a loss of control due to the sudden explosion of fear or anger from an inadequate cause. The interparoxysmal state of the hysterical is characterized often by morbid fears and obsessions and by psychic tics. Thus the psychasthenic, who despite his morbid fears is forced to do things he dreads, will go into an emotional crisis resembling an hysterical attack.

Diagnosis.—The diagnosis of a psychasthenia, characterized by morbid doubts and fears, fixed ideas and morbid impulses, is usually very easy. Sometimes the condition is associated with a great deal of mental depression and anxiety, so that it may be something like a melancholia; but there is with it none of the profound melancholia, with suicidal feelings, and the condition is one more of anxiety and alarm than

of true emotional depression. There is, furthermore, no difficulty or retardation of thought, nor any of the apathy of melancholia. Persons with fixed ideas or obsessions may seem at first to resemble, to a certain extent, paranoiacs, and such cases have been described as abortive types of paranoia. The fixed idea, however, is one which the patient realizes to be over-dominant and in a sense forced upon him; he does not accept it with sympathy and conviction as the paranoiac does; he is aware that it is, in a measure, delusional in character. In the case of morbid impulsive acts, the question of an underlying melancholia or other psychosis must be considered. The splitting up of the various clinical symptoms into special groups of anxiety psychosis, conversion hysteria, true (actual) neuroses, psycho-neuroses is a matter for psychologists. It does not seem to me necessary.

Prognosis.—Psychasthenia associated with morbid fears and doubting manias, occurring in early life, is a serious condition. If taken early in hand, however, and placed under rigid control, the patients have an excellent chance of recovering in one or two years. If, as is often the case, the patients are kept at home and not properly managed, the trouble becomes fixed, some mental deterioration develops, and a serious and incurable condition takes place.

When the disease develops later in life, as an episode or as the result of some shock or exhausting effort, prompt treatment is usually effective; but here again, if the trouble be neglected and becomes fixed, the prognosis is more serious.

Psychasthenic conditions associated with fixed ideas and compulsions are in my experience less serious. They often disappear under tonic treatment and rest in the course of a few months or a year, but there is the liability that they may return.

An obsessive psychosis rarely develops in the degenerative period of life; if it does, it is serious and is apt to turn out to be a melancholia. This outlook is apparently much brighter than that taken by some writers. It is perhaps because neurologists see cases earlier and do not deal greatly with custodial patients.

Treatment.—The treatment of psychasthenia in all its forms is essentially one of hygiene, tonic measures, occupation, restraint and pedagogical training. Without such measures very little can be done; but with them all but the worst cases can be managed with fair satisfaction. This general statement applies to dipsomania and the drug habits, as well as to the obsessions and the morbid fears and doubts. It is as useless to treat these cases (except in the mildest forms) by medication and at home as it is to treat the drink habit or the major hysterical conditions. There are no specific drugs. In the mild types, characterized simply by fixed

ideas and a certain amount of fear and doubts, hypnotic treatment is certainly of some use. This applies especially in the younger cases.

The method of psycho-analysis and mental catharsis, of hunting for the irritative subconscious complex, and explaining the mechanism of the obsession, sometimes cures, sometimes does harm. All the cases thus cured can be relieved by simpler measures, though in so far as psycho-analysis means pedagogics and re-education it is exactly in line with the older and long employed therapeutics.

CHAPTER XXIII

NEURASTHENIA (NERVOUS EXHAUSTION, BEARD'S DISEASE)

The condition which is called neurasthenia is often only an exaggeration or pathological emphasis of a neuropathic constitution. Persons of a highly nervous organization often have at various times, some symptoms of neurasthenia.

Neurasthenia is a functional nervous disorder, characterized by nervous weakness and irritability, so that the patient is exhausted by slight exertions, reacts excessively to irritations and is largely or entirely incapacitated for his daily tasks. In popular usage, neurasthenia is a name often given to what is really some serious disorder such as melancholia.

Nervous exhaustion is rarely a disorder by itself, but is more often a condition associated with some morbid mental state.

Besides or with this general neurasthenic condition there are local neurasthenias, affecting especially vasomotor, gastric, enteric and sexual functions.

There are evidences that the neuropathic constitution existed in all ages, but coherent descriptions of clinical types, like the neurasthenia of modern days, are not found in literature until the past century. The credit of calling attention to this condition most insistently, most acutely, and most successfully is due to Dr. George M. Beard, though he described conditions some of which are now placed in other groups. The evidence is rather conclusive that the human race now suffers relatively more from nervous irritability and exhaustion, in its various types, than it did in the past. We find also that neurasthenia is more frequent among the partly civilized and pioneer races such as that of the United States. One sees it in the white and yellow races, but not in the brown or black. The nervous temperament and the strenuous activity of North Americans are said to be due in part to the waves of cold air which pour down upon our Western and Northwestern States, causing quick changes of temperature and a cool, dry, stimulating climate. Neurasthenia prevails rather more in dry temperate climates, but it is by no means infrequent in the tropical regions, and is to be found in the West Indies and in the republics of South and Central America in its classical forms. White people living in the tropics, unless under the best sanitary conditions, become nervous, and Dr. Woodruff asserts that its cause is the excessive light acting upon non-pigmented skins. Neurasthenia is found rather more often in men than in women, but the difference is not great.

The neurasthenic age ranges from eighteen to fifty-five, but the larger proportion of cases is met with between the years of twenty and fifty. Occasionally symptoms resembling neurasthenia may be seen in children of the age of twelve or thirteen, and occasionally also there develops a kind of senile neurasthenia, which is, however, often associated with hypochondriasis, and some definite degenerative changes in the nervous or vascular system.

In men neurasthenia occurs more often in the single; in women the relation is somewhat reversed, so that, taking both classes, the married and the unmarried are about equal.

Neurasthenia does not much affect the people of the country and small towns, though it does exist there. In great cities the number of neurasthenic women, among the wives of laborers and artisans, is rather large, and this is the natural result of the strain of living with husbands who are dissipated, and of rearing large families of children in the close quarters of a tenement house. The disease is relatively more frequent in the educated classes.

Hereditary influence plays a very considerable part in the development of neurasthenia. We can usually find that there is a history of migraine or some nervous irritability, of alcoholism or some psychosis upon one side or the other. A distinct history of the major neuroses or of severe mental diseases is rare, but there is no doubt that a very large proportion of neurasthenics come into the world with an over-sensitive and weakened nervous system. They may be strong enough to undergo the ordinary strain of life, but break down under some specially exciting cause.

The exciting causes of neurasthenia are very various, but they can most of them be classed under the head of excessive mental strain, shock, sexual abuse, and the influences of exhausting fevers, of chronic infections like syphilis, and of excesses in alcohol.

The practice of masturbation is one of the things for which neurasthenics very often keenly reproach themselves and over which much hypochondriacal brooding develops. Excesses of this kind, however, are usually a sign of a degenerative or unbalanced nervous system rather than a cause. The actual harm done is greatly exaggerated, however strongly this practice is to be reprobated. Excessive and unnatural indulgences, such as sodomy, coitus interuptus, etc., tend to weaken the nervous system and are causal factors of neurasthenia. Bad methods of education combined with excessive study may lead to nervous exhaustion. This is usually seen in ambitious students who are forcing their way under great disadvantages through professional schools and into professional practice. Young women, who are excessively devoted to social indulgences, sometimes break down with nervous exhaustion.

The studies and training of the primary and secondary schools may prepare the way for these catastrophes, but they rarely come before the eighteenth year, and then it is not the hard work of schools but the bad habits of life at the period of education which break down the student. Work and study alone do not cause neurasthenia, but are healthful except to certain persons who are of bad stock and are destined to be neurasthenic at the time of adolescence.

Typical attacks of neurasthenia are undoubtedly brought on by the fright and shock incident to severe injuries or exposure to great danger, as in railroad collisions and other frightful forms of accident. A large proportion of the so-called "traumatic neuroses" are partly forms of neurasthenia; and morbid mental states can in time induce a neurasthenia just as the reverse is true. Neurasthenia can be brought on also by excessive child-bearing, the drain of lactation and domestic trouble, great excesses in eating and drinking, and the strain of hard domestic life and of sickness and nursing. Neurasthenia sometimes follows an acute infection like that of typhoid fever or the grippe. It may also be induced by the infection of syphilis (see Luetic Neurasthenia), although this must not be confused with the neurasthenic symptoms which precede paresis and have an organic basis.

Much weight has been laid upon the importance of eye-strain in producing neurasthenia, and, given a neuropathic constitution, there is no doubt that the defect in the refraction of the eye or in muscular equilibrium may cause, or at least keep up, a neurasthenic state. The same is probably true of severe forms of gastric disturbance, and of disease of the pelvic organs, such as subinvolution, decided displacements, and chronic ovaritis or salpingitis. In men the existence of prostatic irritation, of irritable strictures, and hemorrhoids and fissures may start up neurasthenic symptoms.

The existence in neurasthenics of a tendency to constipation, or what is popularly known as "biliousness," accompanied by a gouty or lithæmic diathesis, has been much dwelt upon, and at one time neurasthenia was thought to be largely the expression of a disturbed state of the metabolism —a phase only of gout or lithæmia. This tendency, however, is rather the result of the weak nerve-centres than the cause, though the two often act in a vicious circle. Prolonged and severe dyspeptic disturbances, especially when associated with atony of the stomach and bowels and the condition known as gastroptosis and enteroptosis, are exciting or maintaining causes. Hyperthyroidism and defects in other internal secretions may cause neurasthenic symptoms.

I would sum up the leading causes of neurasthenia thus:

1. Hereditary nerve instability.
2. Overwork and worry.

3. Severe shocks, with or without injury.
4. Infections.
5. Abuse of stimulants and narcotics.
6. Abuse of sexual functions.
7. Disorder of digestive functions, autoxæmia and glandular disturbances. On the whole, the causes are most often a bad heredity and foolish living.

Symptoms.—The symptoms of neurasthenia are relatively definite, but a great deal of what is mental has been written into the disorder. In other words, the minor psychoses such as hysteria, psychasthenia, hypochondria, abortive melancholia have been often looked upon as neurasthenia. The time has come now, however, when these conditions should be recognized and treated separately, and thus greatly limit the field of the disease. For neurasthenia is, strictly speaking, the name to be applied only to a neurosis—a morbid nervous, not a morbid mental, state.

It will be easier to understand what neurasthenia is by indicating first what it is not; in other words, by excluding certain quite well-known groups of psychic disorders.

First, then, we exclude from the symptomatology of neurasthenia the mild types of recurrent melancholia or, as they are now termed, "the depressive phase of manic-depressive insanity." Second, we exclude the psychosis characterized by a dominant sensory complex. It includes patients who are continually complaining of dysthesias, headaches and head paræsthesias, stomach troubles and various bizarre forms of physical suffering and functional disturbance. The disease is seen in young people and it is only an abortive or early type of the melancholia of involution. It is sometimes called anxiety psychosis. Third, we exclude the psychosis (psychasthenia) characterized by obsessions and fixed ideas, of apprehensions, panics of doubts, abulic states, and psychic tics.

Thus we remove from the realm of neurasthenia all the pure psychoses. These psychoses in mild and abortive types give many of the symptoms of neurasthenia, because they usually have a neurasthenic basis.

Under the head of neurasthenia proper there are two types of a neurosis, one neurasthenia præcox or neurasthenia of adolescence, and second, the neurasthenia acquisita or the neurasthenia produced generally later in life by work, worry, shock, infections, toxins, sexual excesses, and other causes already named. Besides these two types, or associated with them, we find local neurasthenias, affecting especially the heart, vasomotor centres, digestive or sexual organs.

The general symptoms of primary neurasthenia are very much like those of the acquired type, and the following description fairly out-

lines the clinical picture of each. In the præcox type, the asthenia is of a more simple character; there is less evident trouble with special organs, less mental distress and fewer psychic symptoms. The young person gives out all along the line, but the malady is apt to be more persistent, and after apparent cure remissions may occur. It is not a frequent disorder, and it may come on long after the ordinary period, which is about the eighteenth to the twenty-fifth years. Whenever this type of neurasthenia is followed by a serious psychosis, such as melancholia, we may infer that the original attack was really a mild psychosis.

One of the first symptoms is an annoying insomnia. It is usually an early insomnia, *i.e.*, the patient cannot get to sleep till late, and then sleeps badly, frequently awakening. Distressing dreams occur, and the patient wakes unrested and more tired than when he went to bed. The patient complains of a general feeling of mental depression. He can work, but soon gets tired. He can play but it exhausts him so that he cannot sleep or has a headache. All mental and physical effort has to be paid for by periods of rest. He wants to work and to play but he cannot. He is easily irritated at things which before caused him no annoyance, and becomes a source of domestic unrest and unhappiness. He suffers from a number of peculiar sensations which are called "cephalic paræsthesiæ." These are sensations of pressure on the top of the head, or a feeling of constriction around the temples, or a burning spot on the vertex, or tenderness of the scalp. Sometimes he has a sense of weakness or even pain in the back of the neck. All these things are increased by the use of the mind or by bodily exertion, and they are all more common in the acquired types. Headache occurs in perhaps one-half of the cases, the headache being usually either frontal or occipital, or both. It is often very persistent, and in fact a chronic headache, not due to tumor or meningitis or syphilis, is almost invariably of neurasthenic origin. This neurasthenic headache is usually diurnal only, coming on in the morning when the patient wakes up and lasting a good part of the day. It does not often keep him awake at night. In this point it is distinguished from the headaches of syphilis and of meningitis or of tumors. Women suffer from these headaches, and from pains in general, more often than men. They in particular have much pain in the back of the neck and along the spine. This keeps them from walking or being upon their feet, and it may develop into a form of trouble known as "spinal irritation."

The special senses are functionally affected. The patients can often see quite well, but their eyes soon tire; they cannot read a book long because it makes the eyes smart or produces some headache. Examinations of the neurasthenic's eye frequently show the existence of some refractive error, most frequently astigmatism and hypermetropia; de-

fects in the ocular muscles, and especially weakness of the internal recti, often occur. Patients have frequently complained to me of a defect in visual memory. They see a thing or face but do not remember it again as readily as they used to. There is no limitation of the visual field in true neurasthenia uncomplicated by organic disease, but there is a morbid susceptibility to fatigue, particularly of the periphery of the vision, so that after long testing, objects in the periphery becomes less distinct, and a sort of artificial limitation of the field may be produced. In some cases an object which is brought from without into and across the visual field is seen in wider range than an object which is placed in the centre of vision and carried gradually out toward the periphery. This is the reverse of the normal condition, and is known as "Foerster's shifting type." A slight drooping of the lids, and excessive mobility of the iris have been noted in neurasthenia.

Neurasthenics of the acquired type sometimes suffer from tinnitus, which is very distressing and aggravates every other nervous symptom, but this usually occurs only in connection with actual disease of the middle ear, or in old people with degenerative changes in the cerebral blood-vessels. An excessive sensibility to noises, and even the pleasant sounds, like those of music, may be present. Neurasthenics sometimes cannot bear even the most enchanting melodies. A similar morbid sensibility to taste and smell may be present. But these are matters of minor moment and are due to temperamental or psychic complications.

In neurasthenia the general muscular strength is lessened and easily exhausted although the patient may not have any actual atrophy.

The cutaneous and deep reflexes are generally exaggerated. These conditions vary considerably, however, in different cases, and are more marked in the younger patients and those of a neuropathic constitution.

The sexual function is irritable and weak and in certain cases this local trouble dominates the situation. But here a psychosis is often complicating the neurasthenia.

There is a considerable disturbance of the heart function in neurasthenia. The most frequent condition is an acceleration of the pulse beat from very slight cause. A pressure over some painful point in the body will sometimes bring up the pulse from 80 to 90 to over 100, and it will remain there for one or two minutes. This is called "Rumpf's symptom." Arhythmia and palpitation of the heart are less frequently observed. Cardiac weakness is an important condition in many forms of neurasthenia and underlies many of the asthenic symptoms. This is particularly true of the neurasthenias of more advanced life. The blood-pressure is usually low. There is an abnormal difference between the pulse-rate and pressure in the vertical and horizontal positions (Crampton test).

The existence of some degree of thyroidism should be looked out for in patients with very irritable hearts. Ordinary valvular lesions have little to do with causing neurasthenia, but when a patient with a bad myocardium becomes neurasthenic his heart gives him much trouble. He feels its irregular beating and skipping and he hears its throb when he lies on his pillows; its action is disturbed by very slight exertion and slight anginal sensations are present.

A great deal of emphasis has been laid upon the vasomotor disturbances of neurasthenia. The most common symptoms are those due to weak and irritable vasomotor centres, viz., cold hands and feet, flushings, pallor, poor reaction to the cold of the air or the bath. Vago-tonic and sympathetico-tonic seizures are more common in psychasthenia.

Many of the symptoms which we now call "neurasthenic" were described by Dr. Prout and Dr. Golding Bird early in the last century and were held by these gentlemen to be due to oxaluria. This was a condition characterized by flatulent dyspepsia, melancholia, and nervous irritability and was thought to be due to defective metabolism, resulting in the production of an excess of oxalic acid. As a matter of fact the condition of the urine is of not much importance in the symptomatology of neurasthenia, except in so far as it indicates an autotoxæmia, or a deficient excretion of nitrogenous bodies, or evidences of slowed or quickened metabolism.

The urine of neurasthenia is variable in specific gravity, but, on the whole rather low; and the daily amount is below the average. The functional acitivity of the kidney may be lowered and the blood in patients with complicating cardio-renal disorder should be tested for abnormal nitrogenous constituents and for sugar, urinary examinations not being always sufficient.

The digestion of neurasthenics is usually affected, and a large proportion of them are probably treated mainly for their stomach conditions. The neurasthenic has a feeble digestion, the gastric juice being deficient in acid and the gastro-intestinal motility slow. In a certain number a gastroptosis occurs, and the lower curvature drops below the umbilicus. The patient suffers from flatulence, acidity and epigastric discomfort, with anorexia and nervousness. There is atony of the gastric wall, but no very marked change in secretion, and no especial fermentation (Lockwood). It is only in patients who have abused themselves with alcohol or tobacco, or excessive indulgence in sweets, or with ravenous feeding that worse conditions are found. In people of more advanced age, feebleness of digestion is often associated with a more serious relaxation of the stomach and intestinal walls, and a great deal of atony of the whole intestinal tract, with patency of the iliocæcal valve. In these cases, which we find particularly often in women, the prolpase of the in-

testines, stomach, and perhaps of a kidney may cause a great many distressing symptoms. The condition has been described by Glénard under the name of "enteroptosis," and it undoubtedly is an important factor in keeping up the neurasthenia of some women in adult and middle life.

Its investigation has been much helped of late by bismuth feeding and X-ray photographs, and in all persistent neurasthenias with intestinal symptoms there should be a thorough study of the secretory, muscular and anatomical conditions of the gastro-intestinal tract.

Among the most serious, though fortunately rare, symptoms of neurasthenia involving the digestive tract is the condition known as "mucous" enteritis. This trouble generally attacks women rather than men, and usually women between the ages of twenty-five and forty. It comes on after the patient has become exhausted by prolonged domestic cares or fashionable dissipation, or some shock. It is one of the earlier symptoms of the nervous weakness, and begins with abdominal pain, followed by attacks of diarrhœa, in which tubular casts are passed, or portions of such. This diarrhœa is painful, colicky, and alternates with periods of constipation. There is a somewhat spastic condition of the bowel, as though it were irritated and closed down upon the contents of the intestine. The term *mucous enteritis* is not strictly a proper one, since microscopical examinations and autopsical reports show that the substances thrown off are not mucous mainly and that there is no actual inflammation. The casts that are found in the stools are composed principally of albuminous substances, the product apparently of the decomposition or disintegration of the epithelial cells of the intestinal walls. While mucous enteritis sometimes occurs in persons who are profoundly asthenic without any decided neurasthenia, yet, in the great majority of cases, it is a symptom of neurasthenia, and can be successfully treated only on such a basis.

The respirations in neurasthenia are generally normal, but shallow and deficient respiratory expansion sometimes exists. In women particularly I have often found that there was an actual inability properly to expand the chest and inflate the lungs.

The temperature is normal, and a very variable temperature of the skin is simply dependent upon vasomotor instability.

The composition of the blood is often quite normal. Hösslin finds that even in those patients who appear to be anæmic there is a normal amount of hæmoglobin; however, anæmia certainly exists in many cases, and there is no question that the use of iron is often of great benefit.

Variations in the weight of the neurotic often occur. Neurotic patients may gain or lose ten or twenty pounds within a few months. The secretions of the skin are usually increased, and the patient sweats easily and profusely. In other cases of a less irritative type the skin is inclined

to be dry. Its nutritional condition is poor, the hair falls, and, according to Beard, there is a tendency to early decay of the teeth.

Traumatic Neurasthenia, Traumatic Psycho-neurosis.—This condition is nothing else than neurasthenia with a more than ordinary mental element of anxious receptiveness to physical impressions. The following description is in a degree a repetition, but it is given here as it is very nearly the actual record of a typical case.

The patient is in an accident and has suffered from some physical injury, and great mental shock. He is perhaps helped to his home, and his sprains or bruises attended to. He goes to bed and sleeps; he wakes up the next morning feeling not quite so well as usual, but congratulating himself, perhaps, on having gotten off so easily. He resumes his work and finds that he can do it, though with not quite so much ease as usual and he very likely suffers from some pain due to a strain or bruise that he has received. In a few days—almost always within a week—he begins to notice that he is more nervous than usual, that little things irritate him which did not do so before, that his head seems somewhat confused, and that the effort to work is wearying. His sleep is disturbed, and he wakes up in the morning unrefreshed by his night's repose. He becomes somewhat despondent over his condition, and thoughts of paralysis or some other serious ailment annoy him. His head aches, the pain being more or less constant and diffused, and located usually over the forehead or at the back of the neck. He has unpleasant sensations in the head, such as that of constriction or pressure or scalding feelings. His back also is continually painful, and walking increases it. His nervousness becomes more marked, and close examination shows a little, fine tremor in the hands. He has also sometimes creeping sensations over the body or numb feelings in the extremities. He tires very easily. He is emotional, and becomes more despondent as the days go on. Sometimes he has spots before his eyes, noises in his head, or ringing in the ears. Reading is laborious and increases his headache; so also does attention to work. His appetite becomes capricious and his bowels are constipated. He suffers somewhat from flatulency and dyspepsia. His heart palpitates easily, and the pulse is a little accelerated. Sometimes for a few days there is a little weakness about the bladder or irritability of that viscus. His sexual power is diminished. His blood pressure is lower than normal. Very slight excitement produces sweating of the hands or coldness of the extremities. He loses a little flesh. The picture, as may be seen, is very much like that of neurasthenia of the acquired type plus a little more of the physical and sensory complex.

These symptoms may be several weeks in developing, and during this time he may perhaps consult a lawyer about his case. If so, the anxieties of litigation begin to add to and intensify his troubles. He consults a

physician, and the physician finds the subjective symptoms that I have mentioned. Objectively, when examined, the physician will discover that the muscular power is somewhat weakened, that there is a certain amount of the fine tremor perhaps in his hands. The knee-jerks and elbow-jerks are exaggerated; there are tender points along the spine and upon the head. In making him stand with his eyes closed there is a certain amount of static ataxia discovered. The pupils are often dilated and mobile, and examination of the visual field shows sometimes a slight contraction, at other times the "shifting type" already described. In many cases a degree of peripheral retinal anæsthesia will be discovered. The pulse will be found accelerated, and pressure on a tender point may send it up very rapidly; a slight exertion will also accelerate it. There will be something apparent in the physiognomy of the case which shows the man to be in a nervous and asthenic condition. Sometimes the pains from which the patient suffers in the back and the weariness in the limbs are so great that he remains a good deal of the time in bed. In all cases he will assert most positively that he is unable to work or to take that interest in his affairs that he has previously done. In a good many cases there will be added to the foregoing picture a number of symptoms due to some local injury; for example, the arm may have been wrenched or bruised, and the result may be a certain amount of neuritis and weakness or pain in that member; in other cases the back may have been so severely sprained that the typical symptoms of spinal irritation ensue, and this is particularly apt to be the case when women are injured; in other cases, again, the legs may have been hurt to such an extent that a sciatica or some other form of neuralgia develops.

The foregoing symptoms, varying in amount and degree, will last, with little change, for a very long period of time. If the case goes into litigation, there is added the worriment occasioned by having to go through the disturbing experiences of trial by jury so that what was at first mainly a neurosis becomes a psycho-neurosis, or almost a psychosis. In many cases, after the trial has been settled and damages awarded or otherwise, the patient begins to mend, and in a certain proportion of cases he gets completely well. This is not invariably the rule.

Spinal Irritation (Spinal Psycho-neurosis).—Spinal irritation is a form of neurasthenia in which, associated with the general neurasthenic symptoms, are certain special, painful symptoms, related chiefly to the sensory nerves of the spine. These cases have in the past been described under the head of "spinal anæmia" and "hyperæmia." They may develop in traumatic neurasthenia. The patients are usually young women, between the ages of sixteen and twenty-five. The trouble is sometimes brought on by injuries or by a physical over-strain. Sometimes it seems to be associated with a natural weakness of the spinal muscles and a

consequent curvature. Sometimes it follows acute infectious diseases. The patient begins by complaining of pain in the back—usually in the lower part—and also in the back of the neck. These pains occur on standing or walking, or any exertion, and are so severe that the patients, in the course of a few weeks or months, give up attempting to walk about. They get relief and comfort in bed, and so they go there and remain. The pains are of a heavy, aching character, increased until they become very sharp when attempts at movement of the trunk are made. There is a great deal of tenderness to pressure along the spinal processes, some of these processes being much more sensitive than others. The most sensitive points are usually in the back of the neck and the upper dorsal vertebræ, and down in the lumbar region. There is some pain also upon pressure alongside of the spinal processes. Painful points often vary, and even in a single examination the patient may complain, and complain honestly, of different sensitive vertebræ. Pressure on these points does not often bring out visceral symptoms, as the brothers Griffin taught, but may cause faintness, nausea and tears from the pain. The patients suffer much from headaches. The arms are often weak, so that attempting to sew or write or hold a book causes pain in the neck and shoulders. The legs are also weak and the circulation is poor. There is sometimes palpitation of the heart and precordial distress. A certain amount of dyspepsia is always present, and constipation is the rule. The patients often have attacks of vomiting, and attempts to feed them require much care. The menstrual functions become irregular. The patient grows weaker and often becomes bedridden, especially if little attempt is made to overcome the symptoms by voluntary effort and attention to nutrition. These patients generally get well in from one to three years, but occasionally they sink into permanent invalidism. The symptoms are quite as much due to mental sensitiveness and disordered cerebrum as to any local spinal trouble, and the term psycho-neurosis is a more correct one.

Local Neurasthenia.—There are cases in which the specially weak and exhausted function is the heart and vasomotor centres, or the stomach and intestinal tract, or the sexual organs. The symptoms here are much the same locally as those described above under the head of neurasthenia. But the patient with a nervously weak stomach or heart or sexual organs may yet be strong enough to do a fair amount of work. It is only one or two of the somatic functions that are involved. Here the term local neurasthenia is a justifiable one. In many of these cases, however, a mental element develops and often local neurasthenias are also associated with a psychosis.

Angiopathic Neurasthenia.—Here the patient has some general symptoms of neurasthenia, but in particular he has special symptoms

which consist of a sense of pulsation or beating, which involves the whole body. The tension of the pulse is low, the rate normal or slightly accelerated. He does not have palpitations of the heart, as in Basedow's disease, and there is no particular dyspnœa on exertion. The skin usually shows a striking degree of dermography, and there is an epigastric pulsation, as well as pulsation of the carotid. The pathology is suggested by the fact that one of my patients was rather promptly cured by adrenalin. And the trouble may be due to an inadequate activity of the suprarenal capsules.

Neurasthenia Gravis.—In instances which are fortunately very rare neurasthenia assumes a very severe and serious type of exhaustion. The patients suffer from the typical symptoms in much the ordinary way, but the degree of weakness is very much exaggerated. Such patients have not only headaches and disturbed sleep, pains in the back and paræsthesiæ, digestive disturbances, and mental depression, but they speedily emaciate to a considerable extent. They take food in fair amounts, but it gives them no strength. The most careful applications of the "rest cure" secure for them only temporary benefit. They cannot walk far without intense fatigue and exhaustion, with subsequently severe headaches, or even attacks of vomiting and diarrhœa. Despite closest examination, no distinct signs of organic disease can be discovered, and I have known such patients to go on into an invalidism which has lasted for over eight years with final recovery. In these cases there is not an hysterical or even large hypochondriacal element. No amount of suggestion or "mind cure" has much effect upon them. They are not, in other words, hysterical, bedridden women, but often men who have reached or passed the middle period of life. Even these very worst cases may get well.

Pathogeny and Pathology.—Victims of neurasthenia are persons who in all cases have either inherited or acquired a nervous system with lessened power for active functioning. There probably underlies in neurasthenia an impaired metabolic power. The nerve-cells break down too easily and build up too slowly. This defect is partly inherited, but is brought out in the struggles of maturer life. Glandular defects (thyroid, adrenals) may pronote the trouble and so may disorders of the autonomic system and the vasomotor centres. There may be cases of pseudo-neurasthenia in which the exhaustions and irritations are psychogenous. Neurasthenia then becomes a symptom of hysteria.

Diagnosis.—The characteristic of neurasthenia is the easy exhaustibility after mental or bodily effort, and the punishment that follows exertion. The mind is usually alert, there is no profound depression, and thought is not difficult or sluggish at first. There is some constitutional and usually some physical cause for the condition. In all these

ways it differs from the mild abortive melancholias with which it is oftenest confused. The feelings of anxiety and depression, the self-concern are not in great excess of normal.

The so-called "anxiety neurosis" is not a form of actual neurasthenia, but an abortive form of hypochondriacal melancholia. There can be no question of this fact, and the attempt to erect it into a special disease is caused by insufficient experience. I have seen many cases who had attacks of this so-called malady when young develop true melancholia of involution later. The neurasthenic has no undue alarm, he does not tell his story with the voluble iterations of the anxious neurotic; if he does, there is a psychosis imposed on the neural exhaustion.

The beginnings of dementia præcox, mild types of confusional and exhaustion psychoses, early paresis, hysteria, psychasthenia, and malingering are the other conditions which must be noted in making a diagnosis. The diagnostic criteria are given under the description of these disorders.

The diagnosis of the cause involves a thoroughgoing study of the bodily condition, an examination for reflex irritations, for toxæmias, for bad environmental conditions and bad mental training and habits.

A patient may be suffering from a number of bodily ailments, and if this person be at the same time of a somewhat nervous constitution the condition may resemble neurasthenia. Those persons having a very feeble digestion, with dilated stomach and an atonic condition of the alimentary tract, may get depressed, fretful, and sleepless; so a person suffering from some chronic uterine or ovarian or bladder trouble may present many symptoms of nervous irritation. These patients may have only a local neurasthenia or a real local disease with neurasthenia as a reflex condition. It must depend largely upon the good sense of the physician to measure the importance of the local troubles as compared with those of the general symptoms. I believe that the fully developed type of neurasthenia is rarely brought out by local disease alone. Still, I have seen cases with neurasthenic symptoms cured for a time by washing out the stomach, and enormous relief to the nervous irritation to result from treating the condition of the blood or relieving the uterine disturbances.

It should finally be remembered that some neurasthenia usually complicates the minor psychoses.

Course and Prognosis.—There is such a thing as acute neurasthenia. This follows prolonged debauches and long periods of excessive mental strain, with loss of sleep. Such patients may present all the signs of neurasthenia, and get perfectly well in two or three weeks. Neurasthenia, however, is essentially a chronic disease, and when speaking of it we refer to this type of the disorder. It is a disease which comes on as a rule gradually, developing, however, in the course of a few months. It may,

however, come on suddenly after shocks and accidents, and it may develop or follow rapidly after an acute infectious fever. It always reaches its height in a comparatively short time, and runs a course lasting from one or two to seven or eight years. This course is a varying one, and this variation is particularly noticeable when the patient begins to get well. The patient continues to improve for a time and then suddenly falls back, then goes forward again, and thus convalescence progresses. Complete restoration to health is possible and frequent, but the patient always has to take more care of himself than before. As a result of an attack of neurasthenia, men and women who have suffered from it are apt thereafter to lead very saint-like and ascetic lives, and hence they as a rule live long. It used to be said by Dr. Beard that neurasthenics would have a long and happy old age. They pass through the valley of the shadow of death, but the experience may be a profitable, if not a pleasant one.

Neurasthenia præcox has a much more serious prognosis, especially as regards duration. This is the kind that may last seven or eight years, but recovery usually occurs. Acquired neurasthenia often lasts only from six months to two years. Local neurasthenias are also curable, except the sexual type, which is an obstinate affection if it is a purely neurasthenic one.

Treatment.—Naturally, a measure of leading importance in the treatment of neurasthenia is rest, and the problem of how this can be obtained is the first one to confront the physician.

In the severe types of neurasthenia, especially when it occurs in young women, the application of the "rest cure," which has been so ingeniously elaborated and perfected by Dr. Weir Mitchell, is undoubtedly the best treatment. I do not find, however, that men submit themselves readily to this measure, and it seems to me to answer best for those neurasthenic women who suffer also from some hysteria and who are reasonably "suggestible" patients. A modified rest cure can often be secured by making the patient stay in bed until after midday lunch or lie down for an hour after each meal, and go to bed early in the evening. Business men will often cut their business hours down one-half if they are allowed still to continue some work. Experience shows that in most cases a rest is not needed for a very long period; *i.e.*, not for over a few weeks. Some isolation or change may be imperative, but prolonged and absolute rest often is not. The patient is put to bed and kept there for several days, but is soon allowed to sit up for a part of the day, and in one or two weeks may begin to use exercises and enter upon a *work cure*. And it is often part of the program to lead the patient from abnormal passivity to more than ordinary activity. To accomplish this, I often substitute exercise for massage, sending a trainer to the patient, and later sending the patient

to a gymnasium. Many patients do best by putting on a sweater on rising in the morning, exercising vigorously, then taking a cool bath, and then the breakfast. Later the exercise habit is inculcated, and this not strenuously, but in accordance with the constitution and tastes of the patient.

Exercise is done to strengthen the muscles, help the circulation, increase the activity of the skin, and keep the mobility of the joints. But it also should be of a kind to arouse the attention, awaken interest and give a little æsthetic pleasure. This is often a difficult task, especially with women. In its place for them we must use hot boxes the masseuse, the trainer—or domestic work.

Horseback riding is an efficient form of exercise. Many persons are greatly wedded to the exercise of walking, and it seems best to fit their needs. It is, however, a kind of exercise which does not take the patient's mind off himself and does not develop the respiratory functions so well as other measures do. Golfing fills in this lack, and this sport is doubtless of service in neurasthenia. The nurse is a potent feature in the situation. Some patients are almost purely exhaustion cases, and only want rest and feeding and sleep. They are better without a special nurse. In other cases and oftener a suitable nurse who is intelligent, tactful and interesting is a great help.

Change of scene is usually very beneficial to neurasthenics, but traveling is injurious to them. They should be sent to some special place and be made to stay there. A tour along the Mediterranean coast or a trip to Europe often brings them back worse than when they went. Much the same can be said of trips to various places in the South or West. Some of the sanitaria in Germany, some of the places in the Riviera, Egypt and Bermuda, Nantucket, parts of North and South Carolina and Arizona, furnish good resorts for neurasthenics. They generally do better in the mountains, if the altitude is not too high, than they do by the seashore. Dry, windy, sunny climates like those of the Colorado plateau and parts of California and the Northwestern States are too stimulating for most cases.

Much good may be obtained at the numerous sanitaria which exist in this country. Many of these are well conducted, and well supplied with all the modern appliances for treatment. It is, however, always a serious thing to send a neurasthenic to a sanitarium, for the reason that if he stays there too long he becomes contaminated with the atmosphere of invalidism about these places and develops hypochondriacal ideas as to his diet, his liver, his stomach, his sleeplessness, and his various sensory disturbances. In sending a patient to a sanitarium it is a wise plan to tell him not to stay, under any consideration, longer than six weeks; usually four is better. In the summer time great benefit can be secured

by camping out in the woods and living a purely outdoor life, away from the conventionalities and restraints of civilization.

The diet of young neurasthenics should be a nitrogenous one and my directions are that the patient can eat meats, fish, eggs, green vegetables and fruits. Milk can almost always be taken, at least for a short time. The older patients who do best upon milk, vegetables and fruit, with practically no meat. In general, tea and coffee, alcohol and tobacco, are to be entirely prohibited, but this is not an absolute rule. In some cases coffee is beneficial, in some tea does no harm, and in others a small amount of whiskey or dry wine and a cigar are also harmless. The physician has to determine this by the reactions and habits of the patient. Neurasthenics usually drink too little water and it is wise to prescribe a certain amount for them. Four or five glasses of water, which may be either plain or alkalinized, are to be taken daily, unless the patient has a dilated stomach when he should have a dry diet. In dyspeptic patients the meals should be small in amount and taken at frequent intervals; three light regular meals a day and a little food in between form a regimen which usually answers well.

Hydrotherapy, and massage are all measures which prove of service to the neurasthenic. Of these, hydrotherapy is the most useful, though its value can be overestimated. The ordinary prescriptions consist in the cold sponge bath every morning, and, if it is practicable, the use of a Charcot or a Scottish douche every other day. For women wet packs with massage are sometimes helpful, particularly in cases in which there are a great deal of nervousness and motor irritation. At night a lukewarm bath, at a temperature of 95°, for ten minutes, sometimes relieves the sleeplessness.

Massage seems to me of not very much use in men, but it is often grateful and helpful to women, and when a great deal of rest is to be enforced it is essential to employ it for both sexes. Psycho-therapy is always to be more or less consciously applied. The subject has already been discussed and if I do not say more here it is because the methods of DuBois, suggestion, hypnotism, etc., apply much more strictly to the minor psychoses. Neurasthenia proper needs physical attention to the body first, for when it is relieved the psychosis may disappear.

It seemed to me that too much stress is often laid on the importance of minor troubles of the pelvic organs in women. Curetting and sewing up small tears rarely do any good. Serious uterine enlargement and prolapse, displacement and real disease of the ovaries require attention. If, in fact, the disorder of the pelvic organ gives recognized discomfort it should be corrected.

Young men who are distressed by nocturnal emissions and who have

irritable urethras or distended vesicles may require some local treatment. But in these cases there commonly is also a psychosis and there is required more moral than local therapeutics.

The eyes have received and need to receive close attention for they are often even more neurasthenic than the stomach. Attention to errors of refraction and accommodation, and to poor muscular balance is imperative. Poor eyes may keep up the neurasthenia and make the patient suffer from the discomforts of being unable to read or to do close work, as well as give rise to headaches and all kinds of cephalic paræsthesia. But most often the neurasthenia remains after the best oculists have done all that is possible. Many neurasthenics continue so though they have good eyes and can spend their resting hours in continuous reading. This is my experience and my answer to the insistent claims of certain ophthalmologists that profound long continued neurasthenia may be due to poorly corrected vision. Such cases are not seen by neurologists now.

The bromide of sodium or potassium may be given to allay irritability and insomnia; it should be kept up for a limited time and then gradually reduced. At the same time or later the patient may be given a tonic mixture containing such drugs as the symptoms suggest. Quinine must be given carefully, as it causes increase of nervousness in many.

Phosphoric and muriatic acids are the two mineral acids most often of use. These acids are usually better given after meals. The saccharated carbonate of iron or Blaud's pills, if given, should be given generously, *i.e.*, in doses of thirty grains daily.

The foregoing covers in a general way the measures to be used in treating neurasthenics. It is really the treatment of the neuropathic, and the constitutionally weak, also.

THE SEXUAL NEUROSES AND PSYCHOSES

Of the above disorders the neurologist has to deal chiefly with the vicious habits of masturbation (which may, however, be also a manifestation of disease) and the sexual neuroses, spermatorrhœa and impotence.

Masturbation and Spermatorrhœa.—Masturbation is the name given to the vicious habit of artifically exciting the sexual organs. It is very common among boys and less common but present among girls and adult men and women. It is usually only a vice due originally to low associations and teachings among children. In some cases it is a disease or the symptom of a neurotic or insane constitution.

Etiology.—It is most common between the ages of fourteen and eighteen, but may begin earlier. Even infants and very young children some-

times masturbate, usually as the result of some local irritation which leads them to rub the genitals. A tight prepuce, eczema, or worms may lead to the habit, but is it usually taught by a companion. The practice sometimes attacks schools almost like an epidemic, for in every institution a certain per cent. of the boys are sexually precocious or vicious, while the others are ignorant and innocent of the evils of the practice. Masturbation is relatively rare after twenty, but is practised by some throughout life even up to old age.

Symptoms.—Masturbation, as ordinarily practised, leads after a time to a feeling of malaise, mental depression, disinclination to work, study, or to enjoy one's self as before. The appetite is a little impaired, the extremities easily get cold and perspire readily. Peculiar numb feelings are felt in the hands and feet. There are an unnatural nervousness and irritability, and the power of concentrating the mind is a little weakened. The patients often have dilated pupils and hyperæsthetic skin. After a time nocturnal emissions occur. The organs become irritable and slight excitement causes erections. These symptoms may be slightly marked and pass away in a day or two, or until another indulgence occurs.

Masturbation is sometimes done to an extraordinary extent, even daily or twice daily for a considerable time. After a while the young man begins to find that he is not well and realizes that his habit is hurting him. Then if he be sensible and of healthy constitution he stops. Others are frightened out of it by friends or by reading the terrorizing stories printed in quack advertisements and circulars. Sometimes the fright thus caused leads the unhappy youth into a condition of hypochondriasis, which is helped on by the occurrence of nocturnal pollutions and the nervous debility resulting from his past indiscretions. In other cases in which there is a decided neurotic history, a genuine neurasthenia of a sexual type develops and annoys the patient for years.

Masturbation rarely leads to insanity and is oftener a symptom than a cause of such disorder. It is perhaps most often associated with dementia præcox. It is occasionally the cause of epilepsy. When this is the case the convulsive attacks are likely to put on a hysteroid phase and are accompanied by peculiar co-ordinated convulsions and emotional disturbance. Masturbation is the common cause of hystero-epilepsy in women.

Diagnosis.—Many victims of the masturbation habit, who have come to recognize its evils and tried to stop it, develop a hypochondriacal condition, and feel sure that there is something in their faces which reveals to the world their trouble. This is not the case. But there is a certain physiognomy which in a measure characterizes the masturbator to such an extent that an experienced observer can detect it. The pale, pasty

complexion, moist, furtive eye, dilated pupil, listless, restless, and depressed manner, the wet, flabby palms, and hyperæsthetic skin, all help to tell the story. Locally, the penis is often reddened and more or less turgid, the scrotum relaxed, and a varicocele may be present. Examination of the urine may reveal spermatozoa. The urine also is almost always of rather low specific gravity, and contains a great excess of phosphates, both earthy and alkaline.

Treatment.—The patient must be told plainly the necessity of stopping the practice. He must be impressed, but not terrorized. He should be kept out of doors at vigorous physical exercise, for sedentary and solitary work is always bad for such cases. He should be made to take cold-water baths and should sleep on a hard bed with light covering. He had better sleep with some one whose presence may exercise a controlling influence. He should not eat heartily at night, never just before going to bed. And what is still more important, he should not drink before going to bed. Sometimes it is well to have him wakened at an early hour in the morning, when he should empty his bladder; for emissions occur often early in the morning and are promoted by the irritation of a full bladder.

Locally, cold-steel sounds may be introduced and allowed to remain for ten minutes, three or more times a week, or the psychophor or Ultzmann's short catheter may be used. In bad cases with a great deal of prostatic irritation, local applications of nitrate of silver are needed. Internally, a mixture of tinct. opii, tinct. camph., and tinct, lupulin may be given at night, the ingredients being somewhat varied in amount to suit the case. Bromides, chloral, atropine, and salix nigra are also drugs which are often useful. The mechanical measures which have been devised for preventing erections, such as rings with sharp teeth, are rarely needed and rarely useful. They may even do harm by directing the mind to the affected function.

I do not believe it right for the physician to prescribe fornication. It is not safe nor curative, apart from the moral aspect of the matter. It has always struck me also as pretty small business for a man purposely to select a wife to relieve him of the results of a weak will and vicious sensual indulgence. If marriage comes in the natural course of events, as it often does, so much the better. But to select a wife as a remedial agent for masturbation is unjust to the woman and a confession of moral and mental feebleness. Man is distinguished from the brute by his self-control. Let him bear this fact in mind and raise himself above the animals by a determined effort of the will. Pure thoughts and chaste associations, vigorous physical exercise, and a resolute effort to act a manly part will always be successful.

TRAUMATIC NERVOUS AFFECTIONS

(*Traumatic Neuroses and Psychoses, Spinal Concussion*)

The present tendency of neurology is to deny the existence of any special nervous affection produced by trauma or shock. There may follow from these causes:

1. Surgical injuries.
2. Neurasthenic and morbid psychic states.
3. Hysterical states.
4. Hemorrhagic, inflammatory, and degenerative diseases.
5. Combinations of the foregoing.

These troubles may follow not only railway but other injuries, but are especially liable to follow those associated with intense fright.

2. Traumatic neurasthenia or "traumatic neurosis," "railway spine," does not differ from forms of neurasthenia produced by other causes, except that with it there may be certain sprains and surgical troubles and some morbid mental condition. Its special symptoms are described under the head of neurasthenia.

3. Traumatic hysteria is a rare affection in this country. It does not differ from hysteria produced by other causes, except for its sudden onset and occasional surgical complications. It is usually a hysteria major and has the characteristic stigmata of that type. In this city electrical injuries and frights have produced some classical cases of hysteria major.

4. There is considerable evidence that in some rare cases traumatism may produce minute multiple hemorrhages throughout the nervous centres. In such cases there are usually neurasthenic or hysterical symptoms and in addition symptoms of organic disease.

Massive hemorrhages and serious mechanical injury of the nervous centres may be also produced by injury.

Finally, it is a well-known fact that traumatisms may excite, in *the predisposed* and the infected, locomotor ataxia inebriety, insanity or may lead to the development of a cerebral tumor.

It is the mental impression, the shock, much more than the physical injury, which produces the functional neurosis or psychosis.

The symptoms may appear soon after the accident, or, after a period of relative health lasting some weeks the neurosis gradually develops.

The most important practical point in connection with the subject is the diagnosis and the elimination of malingering. This is additionally difficult for the reason that the hopes and anxieties depending upon litigation tend to cause introspection, exaggeration of symptoms and unconscious bias even in the most honest. The opinion among American neurologists tends to favor the reality of traumatic neuroses. While malingering is not rare, yet if the patient has really a traumatic neuras

thenia or hysteria the disease may not be a trifling one. Careful research, however, often tends to elicit the fact that previous to the injury the patient was an alcoholic, syphilitic, or neurotic, and perhaps had already the beginning of his alleged traumatic disorder. In no part of clinical medicine is a careful and searching examination and weighing of symptoms more urgently called for. The methods of carrying out such examinations are given elsewhere. Special methods for testing anæsthesia are sometimes needed. The two sides of the body should be tested simultaneously with concealed needles, beginning on the trunk, or the faradic current with a double-pointed electrode may be used. There are few patients who can successfully deceive in an examination covering all the special senses.

The treatment of these neuroses calls for no special notice here.

CHAPTER XXIV

THE MYOCLONIAS OR TWITCHING SPASMS

(*Chorea—The Spasmodic Tics*)

Myoclonia is, or should be, a general term used for twitching spasms of all kinds. Strictly speaking, it includes chorea, the convulsive tics, paramyoclonus multiplex, certain hysterical spasms, myoclonus epilepsy; also myokimia or fibrillary and wave-like twitchings, and rhythmic myoclonia.

Myotonia, or tonic spasm, is a general term used to designate tonic spasms such as those of tetanus, tetany, the symptomatic tonic spasms of organic disease and hysteria, myotonia congenita or Thomson's disease and myotonia acquisita.

Myoclonus is a special term indicating the twitching of a muscle.

Myotonus indicates the tonic contraction of a muscle. The patient has a myoclonia; the muscle is in myoclonus. The term myoclonia has been much misused, and there is great confusion in the nomenclature.

There are five more or less distinct clinical types, but these can be with sufficient accuracy reduced to three:

1. Infectious myoclonia, which is the *chorea* of Sydenham.
2. Psychogenic and degenerative myoclonias which include the *spasmodic tics*, and hereditary chorea.
3. The *myokimia* disorders, which include various symptomatic fibrillary and wave-like twitchings, and the so-called myoclonus multiplex of Friedreich.

The twitching and tonic spasms and gross tremor often occur in the same disorder and we need terms like myoclonus-tonus and myoclonotonia to indicate these combinations.

CHOREA OF SYDENHAM

(*St. Vitus's Dance*)

This is the common type of chorea, and is the disease ordinarily meant when the term chorea is used. It is a subacute disorder characterized by irregular, inco-ordinate twitching movements. The disease is a common one, forming about one-fifth of the nervous diseases of children.

Etiology.—Most cases occur between the ages of five and fifteen. It is very rare under five. A few cases occur after twenty, and even up

to old age, when a senile chorea is sometimes observed. It affects girls more than boys in the ratio of about 2.5 to 1. In adult life the disproportion is less marked. It is relatively rare in the negro race, especially in those of pure blood (Mitchell). It occurs in all climates. Most cases develop in the spring months, next in the autumn, next in winter, and last in summer. The seasonal influence varies in different localities. School attendance and work have something to do with these variations. The disease is more frequent in cities, and in the poorer classes. Hereditary influence is slight, but it exists. In a small percentage of cases one parent has had chorea, epilepsy, insanity, or a decided neuropathic constitution.

The chief exciting causes are injury and fright, mental worry, and some infection, generally a rheumatic or tonsillar one. Fright or some emotional disturbance is a cause in about one-fifth of the cases. Acute rheumatism is given as a cause in very varying proportions, ranging from 15 to 20 per cent. Some authors lay much stress on rheumatism and, counting attacks of tonsillitis as evidence of rheumatism, they find it a large causal factor. Endocarditis is developed in the course of chorea in about one-fourth of the cases, and this may exist without any other manifestations of rheumatism. Pregnancy is a cause of chorea, generally in primiparæ and in young women under twenty-five. Chorea sometimes follows infectious fevers, especially measles, scarlatina and whooping-cough. Chorea is the prominent symptom in some septic cases, of which septic chorea (Sachs), and the serious mental disorder known as chorea insaniens are examples. Overstudy and the worry of examinations are factors in causing chorea in predisposed and badly nourished children; and anæmia and malnutrition underlie most cases.

Symptoms.—The disease may begin suddenly, but usually it develops slowly, and it is not till one or two weeks that the symptoms are decidedly prominent. It usually begins with irregular twitching of the hand or face on one side. The child winks, grimaces, jerks its head or shoulder, twitches its arms and drops things from its hand. The foot and leg become affected later and the child stumbles in walking. In two or three weeks the opposite side is involved, but usually less than the one originally affected. In three or four weeks the disease reaches its height. The patient's movements are then almost continuous. The hands can hardly be used and the child has to be fed and dressed; even walking is awkward and difficult. Speech is indistinct and confused from the irregular movements of the lips and tongue. An actual mutism may occur. The muscles of respiration may be involved so that the rhythm is uneven.

The choreic movements usually occur both when the muscles are at rest and during volitional acts. In some cases the disease is chiefly characterized by inco-ordinate movements when purposeful acts are

attempted. In other cases voluntary movements can be readily performed, and the muscles twitch only when the limbs are at rest. The movements cease, as a rule, during sleep. But the child sometimes gets to sleep with difficulty on account of the movements. In severe cases attacks of mental excitement and even delirium come on for several successive nights. Apart from such incidents, the mind in chorea is usually dulled, the temper irritable, and the child much harder to manage (Fig. 233).

The appetite is poor and capricious, the tongue coated, and the bowels are often constipated. The nutrition fails a little; there are anæmia and

Fig. 233.—Chorea, showing grimace and shoulder movement. (*Jacobsohn.*)

a tendency to loss of flesh. The eyes present nothing abnormal. Hypermetropia, astigmatism, and muscular insufficiencies exist, but not much more than in other nervous children.

The child is often worse in the morning and improves toward night. Excitement and physical exertion make the movements worse. There is rarely any pain and never anæsthesia or tenderness. The muscles are weak but not actually paralyzed. The deep reflexes are somewhat lessened and the knee-jerk may be abolished. In a good many cases the so-called "tonic reflex" is present. When the patella tendon is struck the foot flies up, and instead of dropping back at once remains up for a second or more; or a second slight reinforcing jerk occurs. The

electrical irritability of the muscles is, as a rule, increased, but there are no qualitative changes. Nocturnal enuresis occasionally occurs. The urine contains an excess of urea and phosphates and at the height of the attack the specific gravity may be increased.

Forms.—Maniacal chorea or chorea insaniens, is characterized by fever, choreic movements and great mental excitement—especially at night, with delirium, hallucinations and delusions. The patient has in effect a toxic or infectious psychosis. After two or three weeks the excitement lessens and the patient becomes dull and apathetic. Such cases usually occur in adult women, and they are often fatal.

Paralytic chorea. In this form one arm or one side of the body becomes rather weak and powerless. A few twitching movements are observed. This form occurs only in children and runs the same course as the spasmodic type.

Chorea of adult life and senile chorea. The disease when it occurs in the second half of life attacks men rather oftener than women; it is of toxic or infectious origin, but not related to rheumatism. There is usually a neurotic family history of chorea. The attack is usually excited by emotional disturbances. It runs much the same course as juvenile chorea, but is rather more apt to become chronic. When it occurs in old men it is called *senile chorea*. This type is not to be confounded with hereditary or Huntington's chorea.

Duration—Relapses.—The disease in this country lasts about ten or twelve weeks, ranging, however, from six weeks to six months. There may be great improvement followed by a relapse, and in this remittent manner the disease may last for years. If it lasts more than six months it should be called *chronic*. Relapses occur in about one-third of the cases and rather oftener in girls. Relapses occur oftenest within a year of the first attack and much oftener in the spring. After three years relapses practically cease. The number of relapses is usually but one, but the disease may recur eight or nine times. Relapses rarely occur in adults except in the chorea of pregnancy.

Pathology.—Chorea has no definite anatomical basis, though the seat of the disease is in the brain. The irritant seems to come from and act first upon the blood-vessels, causing in severe cases intense hyperæmia, with dilatation of vessels, small hemorrhages, and spots of softening. There are infiltration of the perivascular spaces with round cells and swelling and proliferation of the intima of the small arteries. In chronic cases the evidence of active vascular irritation is less, but there are perivascular dilatations and increase of connective tissue. The process suggests a low grade or an initial stage of inflammation. The cause of this is usually an infective micro-organism similar to that causing the rheumatic symp-

toms and the heart lesions. The infection, is sometimes and perhaps commonly due to the *micrococcus viridans*. In a considerable per cent. of cases (90 per cent.—Osler) especially in those of long duration, there are fibrinous deposits on the walls of the heart. The presence of points of irritation in the cortex and its meninges and in the deeper parts excites irregular discharges of nerve force and produces the choreic movements. The interruption of the voluntary nerve impulses by diseased foci makes these movements irregular. In paralytic chorea the pyramidal tract is probably more seriously injured by some single large focus of congestion, exudation, or hemorrhage. Indeed, I have seen a true hemiplegia develop in the midst of an attack. In maniacal chorea the meninges and cortex are more involved.

Diagnosis.—The disease is easily recognized by the peculiar twitching movements. It must be distinguished from convulsive tic, hereditary chorea, hysterical spasms which include saltatory chorea and chorea major. The distinctions are not difficult and are given in connection with the descriptions of these disorders.

Prognosis.—As regards life, the prognosis is very favorable. In this country death from chorea hardly ever occurs in children. It is more fatal in adults, and especially in pregnant women. This is the statement of European writers. I have never seen a mortality and many cases of chorea of pregnancy have been observed in my service at Bellevue Hospital. Nearly all non-fatal cases eventually get well of the chorea.

Treatment.—The most important single factor in treatment is rest. The child should not be allowed to take violent exercise or to have any excitement. In most cases he should be taken from school, and in bad cases he should be kept in bed. In all cases he should be largely isolated and kept quiet. Cold packs should be given once or twice daily, if the movements are violent. Cold sponging or douching daily along the back is often sufficient.

As specific remedies, arsenic should be given in doses of ♏v. of Fowler's solution t.i.d., increased by one or two drops daily to fifteen or twenty drops or even more. If this causes nausea and gastric pain or headache, the dose should be stopped for a day and then resumed, if possible, where it was left off. The bromide of zinc is also an excellent remedy, when there is a hysterical element. Chloral is most useful in violent cases especially when the patient can not sleep.

Hyoscine hydrobromate in doses of gr. $\frac{1}{100}$ is occasionally efficacious. The salicylates are now much used in the acute hospital cases. In chronic and obstinate cases hypodermic injections of the cacodylate of soda may be tried. A most potent remedy in some severe acute cases is the injection of neo-salvarsan.

HEREDITARY CHOREA

(*Huntington's Chorea*)

Hereditary chorea was first described by a Long Island physician, Dr. Waters, in 1842, later by Drs. Gorman and Lyon, and in 1872 by Dr. Huntington to whom most credit for establishing the symptoms is due. The American cases have been observed chiefly in New York, Connecticut, New Jersey, and Pennsylvania. Cases have been reported also from Germany, France and England. The disease begins before thirty and not after fifty; it occurs about equally in males and females. It is almost always directly hereditary, either through father or mother, usually the latter. It begins without known cause by twitchings in the face; the movements then extend to the arms and legs. The movements are very much like those of chorea minor. They are, however, apt to become more jerky and violent; the legs are especially subject to jerky movements, causing difficulty and grotesqueness of gait. A patient of mine used to be frequently thrown out of bed by the movements. They can, however, often be voluntarily controlled for a time. The speech is early affected. There is no paralysis or disturbance of sensibility. The disease is attended by progressive mental deterioration, sometimes by a tendency to melancholia, and usually ends in dementia. Its course is chronic and very slow, lasting ten or twenty years. Post-mortem, chronic pachymeningitis and leptomeningitis with degenerative changes in the cells of the motor cortex have been found.

Electric Chorea is a name sometimes and wrongly given to very violent forms of the ordinary chorea of Sydenham. The term was first applied by Dubini to a peculiar and progressively fatal spasmodic affection which has been observed almost solely in Italy, and which is perhaps of a podagrous or malignant malarial origin. M. Bergeron in 1880 also described an "electric chorea" in which the patients are attacked by sudden rhythmical spasms. This latter disease has a uniformly favorable course. Neither of these diseases resembles true chorea, nor do they have the character of the *tics*.

The term electric chorea, therefore, is one that should be used, if at all, only with a qualifying explanation.

Procursive Chorea, or Dancing Chorea.—Laycock has described as a separate kind of chorea a rhythmical or trochaic form, which he says affects children, principally girls, and shows itself in spasmodic rhythmical contractions or in sudden rotating or procursive movements of the body. This has been called chorea procursive, or chorea festinans, by other writers. In many cases it is accompanied by vertigo, when the condition of the patient is similar to that of a person who has been whirling around a number of times. Such cases always have decidedly hysterical characters, although these procursive attacks may complicate ordinary chorea.

Chorea Major is a manifestation of hysteria, and has been described under that head. It is not a chorea at all.

SPASMODIC TIC

(*Mental Tics, Habit Tics, Hysterical Tics, Local, General and Endemic Tics*)

Spasmodic tic is a disease to which the name of chorea is often, but incorrectly given. It is a very chronic disorder, and shows itself in the form of quick, electric-like spasms of certain groups of muscles or single muscles. With this there occur short tonic and rhythmical movements. The twitching movements differ from those of chorea in that groups of muscles physiologically related are affected and co-ordinated movements result.

1. There are local, habit and psychic tics.
2. Generalized tics with tonic and rhythmical movements.
3. Tics associated with other diseases, *e.g.*, myoclonus epilepsy.
4. Hysterical tics.

1. **Habit, Local and Psychic Tics.**—There are many persons who go through life with some trick of speech, of gesture, or some peculiar grimace. It may be only a shrug of the shoulder, a twitching of the eyes, or a sniff. These various movements are *tics* of the co-ordinate kind. The spasmodic motion is of itself normal, but is inappropriate and misapplied.

Such movements are often seen in children. They sometimes represent abortive attacks of chorea, and sometimes they are the residuum of old attacks. In these tics, a reflex or central irritation often sets up the spasm. This irritation, such, for example, as that of an eye-strain or a nasal disorder is removed, and subsides from consciousness; but the subconscious feeling of it remains and produces a response, such as the grimace or the sniffling.

Closely allied to habit tics are various local tics. Thus when the spasm is localized in the facial nerve, we have a facial or mimic tic (see p. 105). The spasm may become localized even in a single branch of a nerve, as that to the orbicularis, the zygomaticus, the diaphragm, or the tensor tympani. Spasmodic tic sometimes involves the trunk and legs. It may attack the muscles of expiration and the larynx, and then it has been called *chorea of the larynx*.

Spasmodic tics are sometimes accompanied by explosive disturbances of speech. In these cases the patient at the time of the convulsive movement utters some obscene or profane words (*coprolalia*), or involuntarily repeats the last words of the sentence spoken to him (*echolalia*), or spasmodically imitates a gesture made to him (*echokinesis*), or involuntarily exclaims the thought uppermost in his mind, perhaps revealing some secret against his will (*tic de pensée*). These speech-tics may occur without any other motor manifestation, and a tendency to exclamatory tics is not uncommon in ordinary life.

2. **Generalized Tics** (*Degenerative Tics*).—The convulsive movements here take a wide range and affect a number of groups of muscles, producing quick, violent movements of the face, head, extremities and trunk, associated with short tonic spasm of the affected parts.

Generalized tics affect children from birth or develop between the ages of six and sixteen years, and by preference the masculine sex. There is almost always a neurotic and sometimes a hereditary family history. The children are nervous, excitable, often retarded.

The disease begins with attacks of spasmodic movements, affecting generally the head, face and upper extremities first, then perhaps involving the whole body. The movements can be controlled for a time by the will, only to break out with increased violence later. They cease during sleep, which is generally profound.

Degenerative generalized tics are chronic and incurable.

When there is no gross change the brain shows an irregular arrangement and defective number of the cells of the motor cortex. Sometimes the brain is found damaged by infection, or an early cerebral diplegia.

The treatment of tics is dealt with in connection with certain special types, *e.g.*, facial tic, torticollis, writer's cramp. It is essentially hygienic and pedagogic. In the mild forms of habit spasm of children, bromide of zinc in doses of gr. ii. to tolerance is a very efficient drug.

Spasmodic tic may be associated with epilepsy (myoclonus epilepsy). Of this there is a special familial type (Unverricht).

Hysterical or *psychogenous* tics may simulate the local and the degenerative forms. They are not so severe or general in their manifestations and are more apt to be manifestations of a sexual complex, the results of mimicry and a psychological contagion like the dancing manias of former centuries.

The peculiar disorder of the Maine "jumpers," characterized by sudden violent movements on being touched or startled, is a form of endemic hysterical tic. The diseases known as *latah*, occurring in Malay, and *myriachit*, occurring in Siberia and Kamchatka, are also endemic and probably hysterical tics.

FIBRILLARY MYOCLONIA

(*Myoclonus Multiplex of Friedreich*)

The exact nature of this rare disease has been misunderstood. It is probably due to some infection, but in the original case it followed a fright. It occurs mostly in adult males. In the description of the disease one cannot improve on that originally given by Friedreich. He states that it is a peculiar affection of the muscles, especially of the upper and lower extremities, showing itself in the form of short rapid contractions, reappearing at short intervals, which affect individual muscles of the arms and forearms, thighs, trunk and face.

It is not merely fibrillary or fascicular twitchings, but a spasm which involves the whole of the muscle which swells in a mass, and bulges every time, even when the contractions of the muscles were not marked enough to produce a noticeable motor effect on the limbs to be moved. Only at times, at an unusually marked contraction, one notices a slight change in the part to be moved, *e.g.*, during many contractions of the biceps a slight flexion of the forearm or during some of the spasms of the supinator longus a slight supination of the hand.

The spasms, are unrhythmical throughout and varying in extent, even though they are separated from each other only by short intervals. By placing the stethoscope on the affected muscles in the moment of their contraction, a loud and clear sound, resembling the first heart sound in every respect, can be appreciated.

Every muscle shows a complete independence in relation to its individual contractions. Now it was this muscle, now that muscle, which twitches all for itself without any relation to the others, and if it sometimes happens that the contraction of a muscle on one side takes place synchronously with another muscle of the same or other side, or even at times if two symmetrical muscles contract at the same time, it is only an accidental coincidence.

The frequency and intensity with which the contractions of each single muscle ensue are not the same on different days or at different times of the day noticed. During the times of greater agitation there are 40 to 50 contractions per minute in one and the same muscle, while during the hours of greater rest only 10 to 20 contractions could be counted. Only rarely does it happen that the spasm ceases entirely, or almost entirely.

The foregoing is based on Friedreich's description. It corresponds with several cases which I have observed except that sometimes the trunk muscles are greatly involved. It seems to be a toxæmic disorder, causing an irritation of the peripheral motor neuron. J. R. Hunt found hypertrophy of the muscular fibres in one case. Much has been written into the original description and some cases have been classed as hysterical, but that cannot be the pathology of the originally described disease. In one case observed by myself the muscular phenomena were due to a general infection from which death resulted.

MYOTONIA

This is the general term given to tonic muscular spasms, and they may be irregular, rhythmical, localized, or very general in distribution.

The myotonia may be acquired, and it is then called *myotonia acquisita*, or *symptomatica*. Not much is known of this as a definite malady. It occurs sometimes as an *epiphenomenon in progressive muscular dystrophy and myasthenia gravis*. It is a not uncommon symptom of muscular exhaustion or deficient vascular supply.

Thomsen's Disease (*Myotonia Congenita*).—This is a hereditary and family disease characterized by the development of tonic spasms when the patient attempts voluntary movements. The disorder is very rare.

Etiology.—Congenital myotony is practically always hereditary and runs in families. It affects males by preference and develops at the time of adolescence.

Symptoms.—The patient notices that on trying to rise or walk his legs are seized with a painless spasm, which in a few seconds relaxes, but comes on again when the muscular movements have been repeated. If he closes his hands tightly a cramp occurs and he cannot relax the grip. If he shuts his eyes he cannot open them for a moment. The muscles of mastication may be affected, but the extremities are the

FIG. 234.—Myatonia congenita. (*Spiller.*)

parts most involved. The involuntary muscles are spared. The spasms are increased by cold and nervousness; they are lessened by muscular exercise. The muscles are somewhat hypertrophied, and the patient may present the appearance of a very strong man. The actual strength is fair, but less than would seem. The general health may be good, but the patients sometimes show the signs of low vitality in weak digestion, feeble sexual power, and susceptibility to cold.

The electrical excitability of the nerves is normal, that of the muscles is increased, and there is produced a contraction tetanus by both currents. In addition, Erb describes a peculiar reaction produced by a strong stabile galvanic current. It consists in the appearance of wave-like muscular movements passing from cathode to

anode. This is not always present. The mechanical excitability of the muscles is also increased.

Pathology.—The disease is probably a primary muscular dystrophy, a view supported by its occurrence in the wasting dystrophies. There may be, however, a peculiar defect in innervation resulting from a congenital anomaly of the motor tracts. The muscular fibres are found to be hypertrophied, the striations indistinct, and the nuclei increased.

The *diagnosis* is easily made by the characteristic tonic spasms.

The *prognosis* is bad as regards cure, but the disorder does not shorten life.

Treatment.—Dr. Thomsen, who first described the disease, states that active muscular exercise benefits patients. No specific measures are known.

Symptomatic Myotonia and Paramyotonia.—Paramyotonia is the name given to a form of myotonia in which the symptoms deviate somewhat from the typical ones that appear in Thomsen's disease.

Congenital paramyotonia is a family affection, resembling in this respect Thomsen's disease. The muscular rigidity is brought on not by voluntary movements, but by exposure to cold and often very slight degrees of cold. The tonic spasm is a long one and lasts for from a quarter of an hour to several hours. It affects the arms more than the legs. The facial muscles are prone to become rigid. The attacks are followed by some muscular weakness. In congenital paramyotonia the trouble is undoubtedly a primary disturbance of the muscles; in other words, a myopathy.

Symptomatic paramyotonia is noted most characteristically in a certain form of paralysis agitans. Here the patient, when attempting to walk or to rise from the sitting posture, is suddenly seized with an apparent rigidity of the muscles which prevents him from stirring. The trouble is probably more central than muscular. The myotonic condition appears in the progressive muscular dystrophies, and in spastic paralyses of spinal and cerebral origin.

Ataxic paramyotonia is the name given to a disorder characterized by transient spasms like those of Thomsen's disease, associated with distinct ataxia and also with weakness and some anæsthesia (Gowers). This disease is probably located in the spinal cord and should perhaps be considered one of the forms of symptomatic paramyotonia. No special treatment can be given for either of the two latter forms of disease, of which very few examples have been observed.

Hypotonia and Myatonia.—A few cases have been observed in which from birth the muscles have shown a persistent and extreme hypotonia. This forms a condition known as congenital myatonia (Fig. 234). It is thought by some to be a form of dystrophy. Minor degrees of myatonia occur in many people and are not to be regarded as morbid conditions. Hypotonia is more marked generally in the female sex, and it is developed by acrobats and freaks. It exists as one of the symptoms of Mongolian idiocy.

TORTIPELVIS

(*Dystonia Musculorum Deformans*)

Cases of this curious disease have been described by several American neurologists, notably by Dr. J. Fraenkel who called it tortipelvis. Oppenheim has described it under the name if dystonia musculorum deformans. It is a functional myotonic disorder characterized by peculiar tonic spasms of the muscles of the trunk and pelvic girdle, which are brought out especially on attempting to walk. The disease develops in early life, affects both sexes and various races. It is chronic in its course.

When the patient attempts to walk his trunk and thigh muscles are seized with slow, irregular, tonic and sometimes clonic spasms, which bend the body forward, sideways, and twist it. The effect is to produce an awkward and grotesque and so-called "dromedary" gait. The legs also are involved in the spasms, the neck and arms but slightly (Fig. 235). When the patient lies down or sits quietly the spasms cease to an extent, but begin again when he attempts voluntary movements. There are no disturbances of sensation or of the reflexes. The mind is clear.

Fig. 235.—Torti pelvis—Dystonia musculorum. (*Dr. Goodhart.*)

The spasms are much like the myotonia of spasmodic tic. The physiognomy of the disease, its response in a degree to pedagogical treatment, the absence of all evidences of organic disease all suggest that the malady is allied to the spasmodic tics.

TETANUS

Tetanus is an acute or subacute infectious disease characterized by violent and painful tonic spasms with remissions and exacerbations. It is due to infection by the tetanus bacillus. It is called idiopathic or medical tetanus when no open wound is found and traumatic or surgical tetanus when such condition is present. When it attacks infants it is called tetanus neonatorum; when the jaws alone are involved it is called lockjaw, or trismus. A form which affects the face and throat is called head or cephalic tetanus. The idiopathic or medical tetanus is more chronic in its course.

Etiology.—It has a special predilection for newborn children in some countries (West Indies) and to a less extent for puerperal women. It affects males more than females (4 to 1). After the first month of life there is practical immunity till after the tenth year. It then increases in frequency to about thirty. Most cases occur between the fifth and

twenty-fifth year (Anders). It is rare after forty to fifty. It is much more frequent in dark races and in some tropical climates (West Indies, South and Central America). It is more frequent in some regions of the temperate zone than others because of an infected soil. It is more common in hot seasons.

It is caused generally by traumatism, and most often by contusions and penetrating soil infected wounds of the hands and feet, hence its prevalence in times of war. In cephalic tetanus there is traumatism of this extremity. In this country traumatism of the hands on Fourth of July causes many cases.

Symptoms.—The disease sets in from five to fifteen days after infection. It begins with feelings of stiffness in the neck and throat and sometimes with chilly feelings. Gradually tonic spasms develop which involve the trunk and head muscles, causing opisthotonos and other forms of rigid spasm. Trismus, or lockjaw, also occurs. The spasms are attended with intense pain. The patient finally lies in a state of general rigidity interrupted by painful tonic spasms. The muscles are of stony hardness. The jaws are set and the facial muscles are involved, the eyes being partly closed, the corners of the mouth drawn up, causing the characteristic risus sardonicus. There may be profuse sweating; some leukocytosis is present; tissue metabolism is not much changed. Fever is generally present and this may rise as death approaches to 110° or 112°. The disease lasts from two to five weeks. There is evidence of irritation and congestion of the spinal cord and injured nerves, but no special anatomical changes are found. A specific bacillus producing a tetanizing poison has been discovered, the *bacillus tetani*.

The *diagnosis* is based on the characteristic history and the peculiar spasms. In strychnine poisoning there is no initial trismus or epigastric pain. In rabies there is also no trismus, but a respiratory spasm on attempts to swallow.

The *prognosis* is bad. About 80 per cent. of traumatic and 40 per cent. of slow idiopathic cases die.

The *treatment* consists of complete rest and quiet in a dark room and the adminsitration of chloral, bromide, morphine, and physostigma. Successful results from injection of blood-serum of an animal which has had the disease are reported. A tetanus "vaccine" has been obtained and lately used with success. This must be administered both intraspinally and intravenously in maximum doses.

TETANY

(*Tetanilla*)

Tetany is a subacute or chronic spasmodic disorder characterized by intermittent or persistent tonic contractions beginning in the extremities and associated with paræsthesiæ and hyperexcitability of the motor and sensory nerves.

Etiology.—The disease is rare in this country, but relatively common in Europe, especially in Austria. It occurs with frequency during the second, third, and fourth years of life and again at the time of puberty. Its rate of frequency then slowly declines and it is very rare after fifty. It affects males much oftener than females up to the age of twenty; after that the difference disappears. It occurs mostly in the working classes. In infants rickets is often noted. The exciting causes are exhausting influences like diarrhœa, lactation, sepsis, fatigue, mental shock, and fevers; also exposure to cold and wet. Alcoholism, dilatation of the stomach, and intestinal entozoa are also causes. It may be produced artificially by extirpation of the parathyroid gland. The disease sometimes appears as an epidemic.

Symptoms.—Tetany begins sometimes suddenly with symmetrical tonic contractions of the hands; at other times there are at first sensations of numbness, prickling or pain in the extremities, with malaise and perhaps nausea; then spasms begin. The attacks affect first and most the upper extremities. The flexors of the forearm and hand are usually involved; the fingers are flexed at the metacarpo-phalangeal joint and extended at the other joints, and the thumb is adducted, producing the "accoucheur's hand." The forearm may be flexed and the upper arm adducted. The knees and

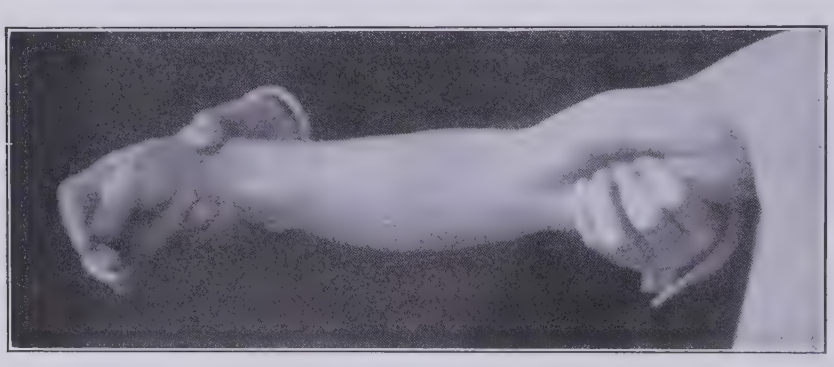


Fig. 236.—The Trousseau symptom.

feet are extended, the toes flexed, and the foot is inverted. In severe cases the muscles of the abdomen, chest, neck, and face are involved. Opisthotonos and dyspnœa may result. The muscles of the face and eyes develop contractions, and trismus sometimes occurs late in the disease. The muscles of the larynx, œsophagus and bladder may be affected. Fibrillary tremors are observed in the contracted muscles. The attacks are accompanied by paræsthesias and cramp-like pains. There may be some abolition of sensation in the skin of the parts affected during attacks. The cramps last from a few minutes to hours or days. They occur during day and night and may wake the patient from sleep. Fever is sometimes present in epidemic cases. The disease has a tendency to recurrence.

While it lasts, both during and between the attacks, peculiar phenomena are observed as follows:

1. *Increased Mechanical Irritability of Motor Nerves.*—The motor nerves show an abnormal irritability, so that on striking the motor point a sharp muscular contraction is brought out. When pressure or a blow is made on the face over or near the exit of the facial nerve from its foramen, contractions of the facial muscles occur, especially those of the lips. This is called the "facial phenomenon." By pressing on the artery and nerve of a limb a tetanic attack can be produced in the muscles supplied.

It is probable that it is the pressure on the nerve alone which causes the phenomenon which is called "Trousseau's symptom" (Fig. 236)

2. *The electrical irritability* of the muscles and nerves is increased, especially to the galvanic current. Thus a negative-pole closure contraction (CaCC) is brought out by a very weak current; and if a little stronger it causes a tonic contraction or cathode-

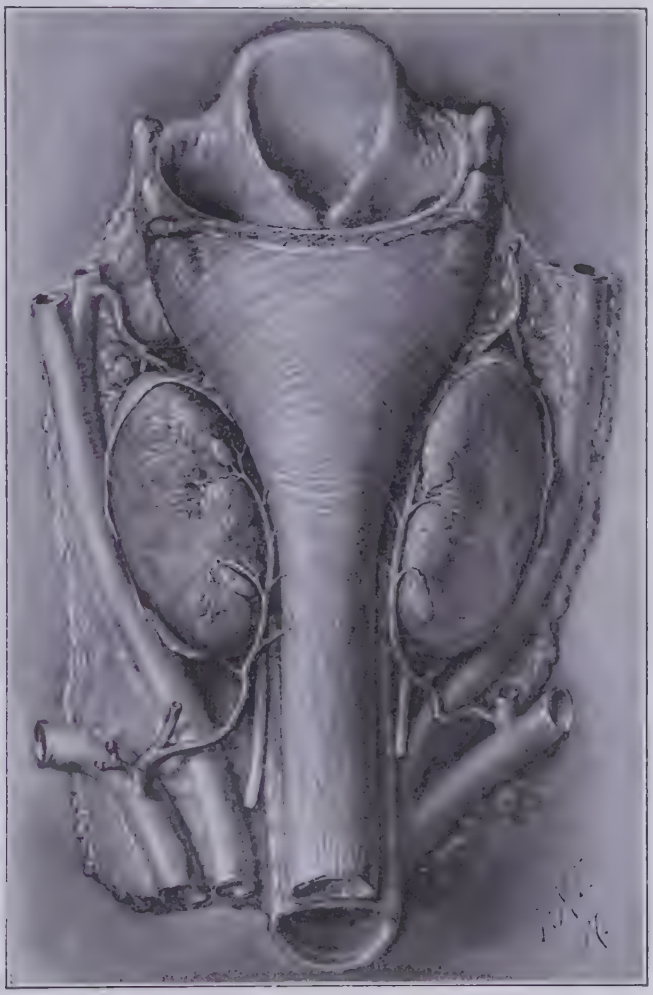


FIG. 237.—The parathyroids. (*Halstead and Evans.*)

closure tetanus (CaCTe). The positive-pole opening contraction (AnOC) may be tetanic, *i.e.*, AnOTe, and there may be even a cathode-opening tetanus (CaOTe), a phenomenon not seen in any other disease. According to Gowers there may be a reversal of the polar formula, so that a positive-pole closure contraction occurs earlier than a negative (AnCC > CaCC). This is certainly rare.

3. *An increase of irritability of the sensory nerves* is shown by pressing upon them, when sensations of prickling and formication appear along their course. There is an increase also in the electrical sensibility, shown by appreciation of very weak galvanic currents. The auditory nerve reacts to the galvanic current in about 15 per cent. of normal cases, and then only to strong currents and to only a partial extent; but in tetany it reacts in nearly all cases, and with comparatively weak currents (2 to 5 or 6 ma.) on anode closure, anode fixed, and anode opening (AnC Klang, AnDKl, AnOKl) (Chvostek).

The phenomena of hyperexcitability above described vary considerably and rapidly during the course of the disease, and are not always present.

Types of the Disease.—The disease varies in intensity and duration. This variation depends much upon the cause, and there have been a number of types of the disease based on the etiology. Thus we have:

1. Epidemic tetany.
2. Asthenic tetany due to lactation, diarrhœa, exhausting diseases, etc.
3. Parathyroid tetany, due to removal of the parathyroid glands.[1]
4. Reflex and toxic tetany from gastric dilatation and intestinal worms.

When the spasms are continuous the disease lasts but a few weeks; when they are intermittent it may continue for months. Epidemic cases last but a few weeks. The disease may be said in general to last from a few weeks to a few months. It is rarely fatal. Patients are liable to a recurrence on return of the exciting cause.

Pathology.—The phenomena of the disease indicate a congested and irritative condition of the gray matter of the spinal cord. The cause of this state is evidently in some cases (epidemic tetany) an infectious poison; in other cases as auto-toxin in the blood (parathyroid tetany), and in other cases it may be a rheumatic or some other toxic influence. Ergot is known to produce symptoms resembling tetany.

In infantile tetany the irritation is apparently due to rickets and the reflex irritation of disordered bowels. It is doubtful if any reflex influence can be invoked in adults. Tetany is a functional disease and the symptomatic expression of a toxæmia. This poison may be of different kinds, hence tetany has a claim to be called a distinct disease simply on clinical grounds. It has no such definite pathology as chorea or epilepsy. In the very few autopsies which have been made no definite organic lesion has been found.

Diagnosis.—The disease is usually easily recognized by the character of the spasms, their symmetrical nature, their course, and the phenomena of hyperexcitability of the muscles and nerves. Trousseau's symptom is found in no other disease. The "facial phenomenon," the peculiar electrical and mechanical irritability of the muscles and nerves, are very rare in other conditions. The sensory irritability, especially that of the acoustic nerve, is also characteristic. From tetanus the disease is distinguished by the intermittency of the contractions, their feebler character, the fact that they begin in the extremities and extend to the trunk, and by the absence of trismus, at least until late in the disease.

Treatment.—The cause should be removed if possible, lactation stopped, diarrhœa and indigestion corrected, the stomach washed out, worms expelled, rickets if present

[1] A distinct form of tetany is due to disease of or removal of the parathyroid glands. It is believed that when tetany follows operations upon the thyroid gland itself, it is due to the removal of one or more of the parathyroids. These glands are, in human beings, very small objects—about half the size of a pea, four in number—the upper pair and the lower pair; and, according to Dr. Halstead, they have a definite vascular supply. In cases of tetany, due to partial or complete removal of these glands, the tetany can be kept under control by feeding the patients with these glands or the nucleoproteid obtained from them.

attended to. Rest, nourishing food, and tonics are indicated. Symptomatically, bromide of potassium in doses of ʒiss. to ʒij. daily with chloral furnishes the surest relief. Hyoscine in doses of gr. $\frac{1}{100}$ may be tried. Inhalation of chloroform or injections of morphine are needed in severe cases. Lukewarm baths may be of service; so also may ice bags to the spine. Parathyroid feeding or injection of the nucleoproteid should be tried.

RABIES AND HYDROPHOBIA

Rabies is an acute specific disease, communicated to man from the lower animals, and characterized by severe and painful tetanic convulsions, paralysis and death. The disease occurs in nearly all parts of the world, and is common throughout the United States. All mammals are susceptible to the disease, and birds also may contract it.

Etiology.—The commonest source of infection is the bite of a dog; but the disease may be caused by the bite of cats, wolves, horses, cattle, and other domestic animals. The bites of wolves are the most dangerous, next those of cats, and next those of dogs. It is not communicated by the bite of a rabid man. Bites upon the face and hands, the exposed parts of the body, are more dangerous on account of the rich nervous supply and from the lack of the protection of the clothing. The disease may be transmitted by saliva upon an abraded surface. About one-sixth of persons who are bitten by rabid dogs, become infected. Men are more susceptible than women, and nearly half of the cases occur in persons under 20. Rather more cases occur during the months from April to September. The infection is contained in the saliva, and is secreted mainly by the parotid. Other glands may contain it, and it may be excreted in the milk. The blood and lymph are never virulent. In the infected person the virus pervades every part of the central nervous system, but is especially concentrated in the medulla. It exists also in the cerebrospinal fluid and large nerve trunks.

The microbe of rabies has not been discovered. Whatever it is, it makes its way into the nervous system, along the nerve trunks. It has an incubation period of about forty days, ranging between twenty and ninety days.

The characteristic histological change in rabies is certain bodies discovered in the nerve-cells by Negri. They are called the Negri bodies. These are not easily affected by external agencies, even by putrefaction, and are easily stained and observed under the microscope. By means of observing these bodies, the diagnosis, after death, of the disease is very easily made.

The Negri bodies are specific of rabies, and they have been thought to be protozoa. This has not been proved, and it is still most probable that the virus of rabies is an ultramicroscopical body which is in some way connected with tissue proteids which form the Negri bodies.

Symptoms.—As a rule, there are no symptoms during the incubation period. When the disease develops, there is some numbness, tingling, and perhaps radiating pain at the point where the bite occurred. The person suffers from nervousness, anxiety and depression; the sleep is disturbed, and then some symptoms referable to the throat appear. There is early a difficulty in swallowing. In a few days the symptoms of mental excitement appear. The patient becomes more restless, nervous and insomnolent. He suffers from thirst, but cannot quench it, because attempts to drink cause a painful spasm of the throat. As a result, while he desires to drink, the sight of water frightens and distresses him. Hence, the name *hydrophobia*. Then he begins to have attacks of convulsions, which affect first the muscles of the neck and throat and then involve the whole body, causing symptoms resembling tetanus. At other times, there are very active co-ordinated convulsions, like those of hysteria. Slight sounds or any mental excitement will bring on an attack. Delirium and maniacal excitement complicate the condition, which lasts from one to three days. A general paralysis follows, and the patient dies in coma, from exhaustion, the temperature rising quite high. In cases where the infection is very severe, the system is overwhelmed by the virus, and the patient dies very quickly with an ascending paralysis which resembles a so-called Landry's paralysis (paralytic rabies).

Diagnosis.—In the early stages of the disease, the mental condition of the patient and the convulsive attacks are very suggestive of acute hysteria. The history of a bite and the person's respiratory spasms and throat spasms will generally exclude this. In tetanus, there is more or less continuous rigid spasm between the attacks; the history of the disease and the longer period of incubation in rabies enable us to distinguish it.

Prognosis.—When the disease comes on, the issue is surely a fatal one. The careful application of the Pasteur treatment, by inoculation, reduces the mortality after bites from about 16 to one-half of 1 per cent.

The **treatment** after the attack comes on is purely symptomatic, and consists in the use of morphine, chloroform and motor depressants, such as curare.

CHAPTER XXV

EXOPHTHALMIC GOITRE (FAJANI'S DISEASE, GRAVES' DISEASE, BASEDOW'S DISEASE) AND HYPERTHYROIDISM

Exophthalmic goitre is a chronic glandular neurosis characterized by easy fatigability, rapid heart beat, enlargement of the thyroid gland, protrusion of the eyeballs, tremor, and various neurasthenic, metabolic and vasomotor symptoms.

Etiology.—Exophthalmic goitre occurs much oftener in women than men (four to one). It is a disease of early adult life, occurring chiefly between fifteen and thirty-five, very rarely in childhood, and never after fifty. It is apparently more common in the Anglo-Saxon race, but is not very frequent in America, at least in the Eastern States. I am informed that it is rather common in the Northern Central States. There is very rarely any direct inheritance of the disease, but the family is often a neuropathic one. As a rule, the patient is of a neurotic temperament. Anæmia and debilitating diseases promote its development. Syphilis is rarely present, and probably not a cause. Goitre and heart disease do not seem to predispose to the real trouble, but a goitre may lead to a symptomatic Graves' disease characterized mainly by cardiac disturbances and perhaps some cervical sympathetic symptoms. The most frequent exciting causes are powerful and depressing emotions and severe physical exertion. Rarer causes are injuries and infectious diseases, such as influenza, measles, scarlet fever, typhoid fever, and pneumonia. It sometimes develops after such infections without any previous nervous strain or shock. I have met no cases following directly upon traumatism.

HYPERTHYROIDISM

The symptoms grouped under the term exophthalmic goitre represent a phase in the pathological history of the thyroid gland. When this gland is atrophied and inactive we have hypothyroidism and myxœdema; when over-active, hyperthyroidism and exophthalmic goitre. Qualitative changes occur in which hyper- and hypo-symptoms are mingled.

Exophthalmic goitre, according to Rogers, may be preceded by hypothyroidism, this may be followed by hyperthyroidism, and finally the fully developed type of exophthalmic goitre appears.

Hyperthyroidism is a not infrequent condition, and is characterized by nervousness, emotional weakness, a kind of hypo-manic restlessness,

insomnia, fatigability, moist skin, tendency to palpitations, and other of the minor vasomotor and secretory symptoms of Graves' disease. The condition may go no farther than such hyperthyroid state, or it may eventuate in the final and more definite disease.

Symptoms.—The disease usually begins gradually, and the first symptom is in most cases rapid heart beat, associated with attacks of palpitation. The patient is nervous, easily fatigued, and has sometimes tremor. The next symptom is enlargement of the thyroid gland, and at about the same time the eyeballs begin to protrude. This order of development does not always take place, and occasionally one or more of the five principal symptoms is not present.

The disease is usually one or two years in developing, the heart symptoms being those which begin first and end last. With the symptoms above mentioned there occur many minor troubles which are more or less characteristic. The patient is usually very nervous and irritable; a distressing insomnia may be present. There is almost uniformly a fine tremor (eight to nine per second) of the hands, less marked in the lower limbs and not present in the face or tongue. The reflexes are exaggerated. There is a tendency at times in walking for the knees suddenly to give way. The patient rarely has neuralgias, but does have burning or feverish sensations and headaches. The skin is rather reddened and moist, and the patient at times sweats profusely. Pigmentation and vitiligo are sometimes seen, and urticaria may develop. The electrical resistance of the body is much diminished, being 800 to 1,500 ohms instead of 2,000 to 3,000. There is sometimes a dermographic skin, as in other neurasthenic states. The respiratory function is weakened and chest expansion often falls below one inch (Fiske-Bryson). Attacks of a persistent watery diarrhœa occur. Anæmia is usually present. There is occasionally polyuria, more rarely glycosuria. A study of the urine shows an increase in general metabolic activity. The menses are irregular and amenorrhœa often exists. A slight rise in temperature may occur.

The blood-pressure varies but eventually is somewhat increased.

The *major symptoms* of the disease are:

Mental and physical fatigue.
Tachycardia.
Goitre.
Exophthalmus.
Tremor.

The *minor symptoms* are:

Nervousness.
Sweating.
Insomnia.
Lessened electrical resistance.

Subjective sensations of heat.
Diarrhœa.
Polyuria.
Increased metabolism.

The sense of fatigue, and the easy fatiguability have been dwelt upon correctly by Rogers, as a fundamental symptom. It is practically the same condition as that found in neurasthenia proper. Next to this and as common, is the symptom of tachycardia.

Symptoms in Detail.—The pulse beats from 100 to 120 per minute usually, but may rise to 160 or even 200. Its rhythm is usually steady; but palpitations occur easily, even without exciting cause. The patient may wake up at night with distressing attacks, something like those of angina pectoris, but the intense pain and sense of impending death are

FIG. 238.—Exophthalmic goitre.

usually absent. The heart is dilated and a systolic murmur is often heard at the base propagated along the arteries. Real organic disease, however, is rare. The arteries are dilated and soft. They pulsate strongly, particularly the carotids. A thrill is sometimes felt over the heart and always over the goitre. The arterial tension is normal or low at first; later it is high.

The thyroid gland is usually enlarged symmetrically; later in the disease the isthmus is affected and the three lobes of the gland stand out prominently (Fig. 238). If only part of the gland is involved it is oftenest the right lobe. A thrill is felt over it and a systolic murmur can be heard. The enlargement of the thyroid is slight or not detectable in a small per cent. of cases.

The bulging of the eyes or exophthalmus is usually bilateral and even.

If one eye is alone or more affected it is the right. The exophthalmus varies much in degree. It is not usually very great, but may be so excessive as to prevent closing of the lids and to expose the insertions of the recti. The eyeball may be slightly enlarged (one-tenth). The exophthalmus is due to the overaction of certain muscular bands which lie in the orbit (Miller's muscle); also to stimulation of the superior tarsal muscle which retracts the upper lid. These muscles are under the influence of cervical sympathetic, and not the autonomic fibres. The orbital space, as the eye is pulled forward, is gradually filled by fat and connective tissue. Hence section of the cervical sympathetic does not lead to more than a slight relief of the exophthalmus. The pupils are normal and vision is not impaired, though myopia occasionally occurs. The fundus and visual field are normal. Paralysis of some of the eye-muscles is a rare complication. Weakness of the internal recti and exophoria are frequent. The inability to converge the eyes is known as *Moebius' symptom*. The lids show certain peculiarities. One of these, known as *Von Graefe's symptom*, consists in the inability of the lid to follow the downward movement of the eyeball. When the patient is told to follow the movement of the finger vertically downward the eyeball moves steadily, but the lid catches, as it were, and refuses to follow or does so in a jerky manner. Another symptom, known as *Stellwag's symptom*, is the widely open eye, showing white around the iris with infrequency in winking, especially of the upper lid. A tremor of the lids sometimes occurs.

Course.—The disease progresses slowly. After a year or two it often becomes stationary for a long time. Cases of gradual spontaneous recovery occur. The natural duration of most recoverable cases is two or four years. In those which do not recover, the disease lasts five, ten, or more years; the patient emaciates, the heart becomes weaker, albuminuria and dropsy appear, diarrhœa sets in, and the patient dies of exhaustion or is carried off by phthisis or some intercurrent disease. Other cases, having improved up to a certain point, remain in this state for years.

Complications.—Mental derangement occasionally occurs in the later stages of the disease. Hysterical crises, epileptic attacks, choreic movements, paralysis of the ocular muscles, muscular atrophy, paralysis agitans, Addison's disease, diabetes, locomotor ataxia, and local œdema have all been observed. With the exception of hysterical attacks, these complications are rare.

Abortive Forms.—This name is applied to cases in which only a part of the distinctive symptoms develop. It generally means hyperthyroidism with some of the major symptoms of exophthalmic disease. Tachycardia always exists; with it are tremor and moist skin, lessened

electrical resistance and nervousness. Or tachycardia and goitre may alone be present.

Physiology.—The bio-chemical studies of the thyroid shows that it develops certain peculiar iodinized proteids which are characteristic of its secretory activity. The exact nature of the substances is not perfectly known. The action of the thyroid is to stimulate metabolism, and indirectly to promote certain functional activities. Its secretion seems necessary for the growth and development of all the tissues. The thyroid has a certain interrelation with the pancreas, adrenals, thymus and pituitary.

Pathological Anatomy.—Post-mortem examination of the thyroid gland shows that it undergoes a true hypertrophy with increase of vascularity and of the glandular structure, and increase of colloid material in the alveoli. After the hypertrophy has reached a certain stage, the glandular epithelium degenerates and breaks down, filling the acini with degenerated cells. Later still there may be atrophy and fibrosis with sclerosis of the vessels, hemorrhages and cyst formation.

In the nerve-centres the changes which have been found are small hemorrhages in the medulla and degenerated nerve-cells. In one case of about a year's standing, I found a very marked pigmentation and vacuolization of the cells of the vagus and glossopharyngeal nuclei. In another case of six months' standing, no marked changes could be seen in these areas, but there was a spot of softening at the junction of the pons and the cerebral peduncle. This was ante-mortem and had led to crossed paralysis just a few days before death. In other cases congestion and small hemorrhages in the medulla have been found. In still other cases there were no lesions, so that an actual anatomical change in the nervous centres cannot be said to be established. The heart is dilated and enlarged; endocarditis is sometimes present, oftener not; the arteries are dilated.

Pathology.—Some writers now consider this disease primarily one due to a disordered function of the thyroid gland. This is not entirely the case. Graves' disease occurs in neuropathic persons. It is often associated with a neuropathic family history and often neuroses complicate the thyroid trouble. The exciting and fostering causes are often mental and nervous strain. While the symptoms of the disease, the nervousness, the insomnia, and the vasomotor disturbances are due to the hypersecretion of thyroid, the morbid disturbance often may well be fundamentally a loss of central nervous control. If the nervous centres can be kept quiet long enough, the demand on their part for this excess of thyroid juice gradually ceases and the patient gets well. This is the rationale of the prolonged rest which is the real and specific cure for the disease in its ordinary phases.

Prognosis.—About one-fifth of the cases get well or practically well. Probably over half the cases, if they can be properly treated, reach a fairly comfortable condition of improvement. The cases in which symptoms come on quickly have the most favorable prognosis. In those with marked exophthalmus and goitre the prognosis is not so good. The duration of the disease in recovering cases is from two to eight years. A later development of hypo-thyroidism and myxœdema is not in accord with my observation though it may occur. I have recently seen three now healthy patients who had Graves' disease over twenty years ago.

Diagnosis.—The disease can be distinguished by the persistent tachycardia, with goitre or exophthalmus, and in its early stage by the tachycardia with tremor, moist skin, sensations of heat, nervousness, emaciation, insomnia, lessened respiratory expansion, and electrical resistance.

A symptomatic Graves' disease may sometimes be caused by a goitre pressing on the vagus or sympathetic and causing irregular heart beat and perhaps exophthalmus. In these cases the history of a long-standing goitre exists, the heart's action is irregular, the exophthalmus is usually partial and one-sided. In abortive forms it is necessary to have tachycardia and at least one other of the four major symptoms to make a diagnosis of Graves disease.

Treatment.—Rest is the most important single thing. The patients sometimes need to be put to bed or kept on the back for one to three months. Sometimes a half-rest is enough, with freedom from excitement and worry. A sea voyage can be worked in with advantage. No special diet is needed, nor do climatic influences or baths or mineral waters have much effect, except in so far as they secure a quieter and better environment.

The drugs used are numerous. Of these, the phosphates, the lime salts, iodine, strophanthus, aconite, belladonna, bromides, and iron have served best. In certain very mild cases with small goitres, thyroid extract in small doses does good, but as a rule it is harmful.

Beebe and Rogers have obtained an anti-thyroid serum which, in acute and serious cases where there is great thyroid toxæmia, acts successfully. In the more chronic and quiescent cases it is not so efficient. Beebe has recently prepared another serum which is given hypodermically. A serum from thyroidectomized goats has given some results.

The surgical treatment of exophthalmic goitre is not always successful; and it sometimes happens that symptoms of hypothyroidism develop later. It is best to get the patient into a relatively quiescent state by medical treatment and rest before operating. And it is advisable usually to begin with ligation of arteries in order to see whether this may not be sufficient

before any of the gland is removed. According to Rogers the ligation under local anæsthesia of one or more of the thyroid vessels or the removal of one-half or more of the thyroid gland yields about 50 per cent. of cures. Ligature of the arteries is safer but not so immediately efficient as thyroidectomy. If the patient needs for economic reasons prompt relief, with a certain attendant risk, he should try surgery, at least till we have better medical measures than we have at present. Surgery, or at least thyroidectomy, is dangerous in actively progressive cases and in those associated with a psychosis.[1]

The X-ray and radium are said to have value, and I have seen the latter accomplish results.

For the palpitations, the ice bag placed over the heart and neck is helpful and may be used systematically.

[1] S. Landström's statistics of cases of himself, Kocher, Mayo, Riedel gave cure or improvement in about 80 per cent.; death in about 10 per cent. Landström in 38 cases reports cure in 48.7 per cent., improvement in 17.1 per cent., failure in 26.8 per cent., death in 7.4 per cent. Mayo's figures on cures or improvement 87.8 per cent., death, 12.2 per cent.

CHAPTER XXVI

PROFESSIONAL NEUROSES, OCCUPATION NEUROSES

(Writer's Cramp and Allied Affections)

Writer's cramp is a nervous affection characterized by spasmodic, tremulous, or paralytic disturbances, occurring when the act of writing is attempted. Since the introduction of the typewriter, writer's cramp has become less common. In its place we have, with relative less frequency, stenographer's and typewriter's cramps.

Etiology.—It is a disease of modern times, and has been particularly noted since the introduction of steel pens, about the year 1820. A neuropathic constitution is often present, and sometimes there is a hereditary history. Men are much more subject to the disease than women. The most susceptible age is between twenty-five and forty. It rarely occurs after fifty or before twenty. Clerks and professional writers are naturally much more subject to the disease. Excessive worry, intemperance, and all debilitating influences predispose to it. The chief exciting cause is excessive writing. But this is not all. The writing that is done under strain or a desire to finish a set task is the harmful thing. The style of writing is also an important factor. Writing done in a cramped posture with movements of the finger alone or with the little finger or wrist resting on the table is most injurious. Free-hand writing done from the shoulder according to the American system is least harmful. Shaded or heavy writing with sharp steel pens is also productive of harm. Copying is much more harmful than composing. Authors seldom have writer's cramp. The performance under strain of some specially prolonged task is the most common exciting cause. Injury and exposure to wet and cold occasionally start the trouble.

Symptoms.—Writer's cramp very rarely attacks a person suddenly. The patient first notices a certain amount of stiffness occurring at times in the fingers, or the pen is carried with some uncertainty and jerky movements are made. He feels a sensation of fatigue in the hand and arm, and this may amount to an actual tired pain. The first symptoms may last for months or even years. The hand is rested as much as possible; new pens or penholders and new modes of holding it are tried. Often the patient, fearing the onset of the cramp, and as its result loss of employment, becomes anxious, worried, and mentally

depressed. Sometimes the trouble is worse when beginning a daily task, and it gradually wears off in a few hours. At other times exactly the reverse is the case. When the disease has reached its highest stage, writing becomes almost or entirely impossible. The moment the pen is taken in the hand and an attempt at using it made, spasmodic contractions of some of the fingers, or even of the arm, occur, the pen flies in any direction, and it is impossible to control or co-ordinate the movements. The rule is that, although writing cannot be done, all other complex movements are performed as well as ever. Thus the sufferer from writer's cramp may be able to play the piano, or paint, or thread a needle, or use the hand in any complex movements. This limitation, however, is not always present. Telegraphers, who use to some extent the same muscles as in writing, and who also often have to do a great deal of writing, are liable to suffer from both writer's and telegrapher's cramp at the same time. No evidences of actual paralysis are present in the affected muscles and there is rarely anæsthesia, but the arm aches and is sometimes tender. Sensations of numbness and prickling are present and occasionally a complicating neuritis develops. In rare cases vasomotor disturbances are observed. There are sometimes associated muscular movements of the other arm or of the neck.

The various symptoms occur with different degrees of prominence, so that the disease has been classed under the heads of (1) the spastic, (2) the neuralgic or sensory, (3) the tremulous, and (4) the paralytic forms. These forms are, however, often more or less mixed.

1. The spastic form is undoubtedly the most common, and it has given to the disease its name. Cramp of some muscle or muscles is present in over half of the cases. The muscles of the thumb and first three fingers are oftenest affected, and in some cases the flexors, in some the extensors, are chiefly involved. In telegrapher's cramp it is the extensors, but in writer's cramp the flexors, that are mainly attacked.

2. The neuralgic form resembles the spastic plus sensations of fatigue and pain, which are quite severe and are brought on by writing. There may be tenderness over the arm nerves also.

3. The tremulous type, though rare, is very characteristic. The patient when attempting to write develops a tremulous movement of his hand and arm. This ceases when his attempts to write cease. The tremor usually affects most the fingers used in pen prehension, but it also spreads to the forearm and may even involve the entire extremity. Although this tremor develops on attempting to write, it does not develop on other voluntary movements.

4. The paralytic form, or that type in which muscular feebleness is the dominant symptom, is also rare. In the typical paralytic form the patient, as soon as he begins to write, feels an overpowering sense of weak-

ness and fatigue in the fingers and arm. The fingers themselves loosen their grip and the pen may drop from the hand.

Associated Symptoms.—Writer's cramp is essentially a motor neurosis, and its leading symptom is the impairment of a motor function. Other symptoms, however, both general and local, are always associated with it. These are mainly (1) psychical and (2) sensory, more rarely (3) vasomotor and (4) trophic.

1. *Psychical Symptoms.*—The patient is often nervous, emotional, and mentally depressed at times. He suffers from insomnia and vertigo. Patients are generally unwilling to admit that there is any other trouble than the local one, and only careful examination may bring evidence of constitutional trouble. There are cases of purely mental writer's cramp.

2. *Sensory Troubles.*—These consist of pains, sense of fatigue, feelings of numbness, prickling, pressure, weight, tension, constriction, etc. Hyperæsthesia and, more rarely, anæsthesia are also observed. The most common sensory symptom is that of aching and fatigue, and this is usually confined to the arm, and oftenest runs along the course of the radial and median nerves. The cervical vertebræ may be tender and sometimes patients have a headache in the parietal region of the side opposite the affected arm.

3. *Vasomotor, Trophic, and Secretory Disturbances.*—The condition known as *digiti mortui* has been observed, coming on paroxysmally. In acute cases of writer's cramp with associated neuritis decided vascular changes may occur, such as passive congestion of the hand and arm, with swelling and turgescence of the fingers, and a sensation of throbbing. In bad cases the fingers will look as if they had chilblains.

Electrical Reactions.—Ordinary tests will, as a rule, reveal very little change. Sometimes there is a quantitative increase, sometimes a decrease, of irritability to both forms of current.

Pathology.—Neuritis is undoubtedly present in some forms of writer's cramp, so called. It is not present, however, so far as external tests go, in the typical neurosis. Nor are there any post-mortem observations throwing light on the anatomy of the disease. We must believe, therefore, that it is a neurosis having no appreciable anatomical basis.

While writer's cramp is often complicated with some neuritic disturbance leading to symptoms in the affected arm of pain, paralysis, tenderness over nerves, vasomotor disturbances, etc., there can be no doubt that the lesion in typical cases is central and involves the higher reflex centres and indirect motor and sensory paths. Little more can be said of the pathology than that it is an "exhaustion neurosis," and that its mechanism is allied to the spasmodic tics and especially to stuttering. The same is true of all the other forms of occupation

neuroses, and nothing need be said upon this point regarding them when they come to be considered.

The **diagnosis** of well-marked cases of writer's cramp presents no difficulty. In the earlier stages, however, it may be confounded with a large number of disorders, viz., post-hemiplegic chorea, hemiataxia, paralysis agitans, progressive muscular atrophy, progressive locomotor ataxia, various forms of tremor, lead paralysis, rheumatoid arthritis, neuritis, cerebral and nerve tumors, and tenosynovitis.

In many of these cases it is only necessary to bear in mind the history of the disease in order at once to reach a safe conclusion as to its nature.

If there is a great deal of pain in the arm, with tenderness along the course of the nerves; if there is decided change in the electrical reactions; if there are sensations of tingling, numbness, etc.; and if the patient shows an absolute loss of power in the various groups of muscles, with some incapacity for doing other acts besides the one with which he is specially concerned, then the trouble is undoubtedly peripheral and due largely to an underlying neuritis. The prognosis in these cases is much more favorable. If, on the other hand, the disorder comes on in persons who have done an excessive amount of writing; if it is associated with nerve strain; if the electrical reactions are but slightly changed, the sensory symptoms slight and the motor incoordination is marked, limited to the special class of work, and not accompanied with absolute paresis, the disorder is central and needs both a different treatment and prognosis.

Course and Duration.—Writers' cramp is a chronic disease. It begins insidiously and attacks one group of muscles after another as each is brought into play by new methods of writing. If the left hand is used, that, too, is liable to become affected. The course varies, however; for a time progress may be arrested or improvement set in. When the disease becomes well established it will most often last a lifetime.

Prognosis.—The prognosis is unfavorable, yet not so much so as was once thought. Undoubted cases of complete recovery have been reported, even under unfavorable conditions. The prognosis is much more favorable if the patient begins treatment early and before marked spastic symptoms are present. It is more favorable in the neuralgic forms. Some patients who suffer from a mild form of the trouble manage, by the help of instruments or special pens, to do their work for years. The more acute the disease and the more evidently peripheral and neuritic its origin, the better the prognosis. In over one-fourth of the cases, patients who use their sound arm will not be affected in it.

The facts stated regarding the cause, physiology, and general symptomatology of writers' cramp apply to the other forms of occupation neuroses. A few special details, however, will be given regarding these.

The most common and important are musicians' cramp and telegraphers' cramp.

Musicians' Cramp.—Under this head we include pianists' cramp, violinists' cramp, flutists' cramp, and the cramp of clarionet players.

Pianists' cramp occurs usually in young women who are studying to become professionals or who are especially hard working and ambitious. The absurd "Stuttgart method" of teaching the piano, in which the motions are confined as much as possible to the fingers, predisposes especially to this disease. The symptoms are those of fatigue, pain, and weakness. The pains are of an aching character. They are felt in the forearm especially, but extend up the arm and between the shoulders. Spasmodic symptoms are rare. The right hand is oftener affected, but both hands eventually become involved.

Violinists' cramp may attack the right hand which holds the bow or the left hand which fingers the strings, but more often the left hand is affected.

Clarionet players sometimes suffer from cramp of the tongue and of the laryngeal muscles.

Flute players suffer not very infrequently from slight laryngeal spasms. A similar trouble affects elocutionists. The term mogophonia is applied to this type.

Telegraphers' cramp affects especially those operators using the Morse system, which is still the one most widely in vogue. Contrary to the opinions of previous writers, Dr. Lewis believes that this neurosis is not a rare one and is destined to become more frequent. In this city the cramp is not rare, the proportion being about one in every two hundred. The technical name among operators for the cramp is "loss of the grip." In telegraphing, the extensors of the wrist and fingers are called most into play, and hence are most and earliest affected. The symptoms come on very slowly, the thumb and index-finger being first affected. The victim finds that he cannot depress the key on account of spasm in these muscles, and he finds most difficulty in making the dot characters, such as h (. . . .), or p (. . . . .), or z (. . . .). When the flexors are most affected, the key is depressed with undue force and a dash is made instead of a dot. Sufferers from the "loss of grip" generally have writers' cramp also. While spasm is usually present, the disease may show itself simply in pain, paresis, and incapacity to co-ordinate the muscles.

In *sewing-spasm*, which affects tailors, seamstresses, and shoemakers, clonic and tonic spasms attack the muscles of the hands on attempting to use them in the regular work. Tailors who sit crosslegged sometimes suffer from a peculiar spasm on assuming this position.

Smiths' spasm, or *"hephæstic hemiplegia"* appears to have been observed only by Duchenne and Dr. Frank Smith. It occurs in persons engaged in pen-blade manufacturing, saw straightening, razor-blade striking, scissors making, file forging, etc. In doing this work they have to use a light or heavy hammer, with which strokes are delivered very rapidly and carefully. After a time spasmodic movements occur in the arm used, and the arm falls powerless. As in the cases reported, there are generally hemiplegic symptoms, and also neuralgias, vertigo, and other cerebral troubles, the disease cannot be a purely functional neurosis.

Drivers' spasm has been observed in veterinary surgeons by Dr. Samuel Wilkes.

Milkers' spasm is an extremely rare affection, which was first described by Basedow and seems to occur in milkmaids, never in milkmen.

Cigarmakers' cramp is very rare and often associated with some neuritis.

Watchmakers' cramp and *photographers' cramp* are also to be regarded merely as pathological curiosities.

Ballet-Dancers' cramp.—Under this name certain painful and paralytic troubles occurring in ballet dancers, especially premières danseuses, have been described by Schultz, Onimus, and Kraussold. It does not appear that the trouble is really a co-ordinative functional one, but is rather neuralgic or the result of local strain upon the parts.

The list of professional neuroses is made to include, besides those above given, cramps and co-ordinative troubles affecting artificial-flower makers, billiard players, dentists, hide dressers, drummers, electrical instrument makers, stampers, turners, sewing-machine girls, money counters, weavers, painters and pedestrians.

Prophylaxis and Treatment.—The introduction of typewriters, gold pens, and improved penholders has prevented somewhat the increase of writers' cramp. Stenographers rarely have it unless they write in long hand also. Persons who have to write a great deal should use large cork or rubber penholders and gold or quill pens with smooth paper. The best style of writing is that done from the shoulder, but

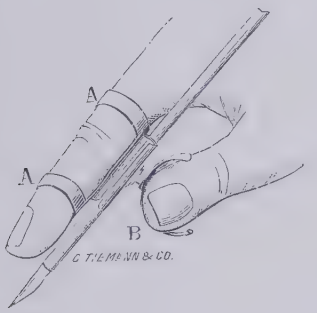


FIG. 239.—Mathieu's instrument for writers' cramp.

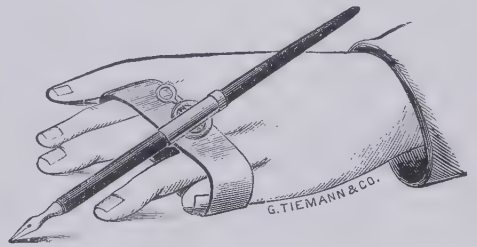


FIG. 240.—Nussbaum's instrument.

this is a method that bookkeepers and those who have to keep accounts cannot easily adopt. Many nervous persons have a bad habit of gripping the pen very tightly and pressing down on the paper with excessive force. Fatigue soon results and painful sensations develop in the arm. Proper attention should be paid to the position of the paper written upon, the height of the desk, the light, and the sleeves of the coat or dress. The paper should be laid at an oblique angle to the edge of the desk, and not at a right angle as many writing-teachers are accustomed to direct. As some cases of "cramp" are undoubtedly cerebral, it is very unwise to attempt any extraordinary exploits in writing or to work with the ambition to put the writing-capacity to the utmost test.

When the cramp is fully developed, the most essential thing is rest, and it is generally best to advise the patient to change his occupation at once. Some rest, however, may be secured by getting a new form of penholder, holding the pen in a different way, using the unaffected arm, or using some form of mechanical appliance. The mechanical

appliances are splints, rubber bands around the wrist, and various instruments contrived to prevent spasm and throw the work of writing on new and larger groups of muscles.

Instruments for writers' cramp are very numerous. Those that are of some value are Mathieu's, Nussbaum's, and some modification of Cazenave's (see Figs. 239, 240). All the various instruments have been of service, or have even been curative in some special cases, but not too much must be expected of them. As a rule, they are only palliative. A cheap instrument that may prove satisfactory is that of Mathieu.

In the medical treatment of writers' cramp the important agents are massage, pedagogics, and rest.

CHAPTER XXVII

PARALYSIS AGITANS

(Shaking Palsy, Parkinson's Disease)

Paralysis agitans is a chronic progressive disease, characterized by tremor, muscular rigidity and weakness, and by a peculiar attitude and gait, together with sensations of heat, pain, and restlessness.

Etiology.—The disease occurs more often among men than women in the proportion almost exactly of 2 to 1 (155 males, 82 females). More than two-thirds of the cases occur between the ages of 45 and 60, and the serious decade is the fifth. Practically no cases occur before 35 and very few before 40.

In 127 personal cases the age of development of the disease was:

20–30,	2
30–40,	8
40–50,	40
50–60,	46
60–70,	24
70–80,	7
	127

The disease is by no means rare in this country and affects the native stock seriously. Among 90 private cases 47 were of distinctly American stock, 13 occurred among Hebrews, and 8 among Germans, 8 among Irish. It is not especially an urban disease, many cases coming from small towns. It affects educated and professional classes more than it does laboring men. Fifteen per cent. of my private cases were lawyers, doctors and clergymen, one was an engineer and one a teacher. It is a disease of brain-workers more than muscle-workers.

Hereditary influence has some importance. Among 90 cases there was a history of apoplexy, heart or renal disease in the direct ascendants in 19 cases, showing a weakness in the direction of the circulatory system. There was a direct heredity of paralysis agitans in 2, an indirect (paternal uncle and aunt) in 2, and a family or senile tremor among 8. Thus about 15 per cent. show a hereditary weakness as regards tremor. Melancholia was present in the ascendants of 7 cases, and a tendency to anxious depression is very common among the patients

themselves, three of whom had the melancholia of involution. Diabetes was present in the ascendants in 2 cases and in 3 of the patients themselves. Not many families, aside from those referred to, were neurotic or rheumatic, and the children of the patients were almost uniformly reported healthy. Some hereditary influence thus is present in 10 to 20 per cent., shown mainly in a tendency to benign tremor, stuttering or a tic, or in arterial sclerosis leading to cardiac disease and cerebral hemorrhage. Paralysis agitans is distinctly a disease of sober living. It is not caused by the ordinary departures from rectitude, such as dissipation, sexual, alcoholic, tobacco or food excess or even by lues. The thing most commonly observed is a regular laborious and anxious life. The patient, if a man, has not been a vacation man, and his habits have been, if anything, abnormally regular. Among direct causes, there can be no doubt that work and worry and psychic trauma are the most important.

Fig. 241.—The attitude in paralysis agitans.

Shock and injury are noted in 2 per cent. of cases, but the influence is not important in all of them. In the working classes, exposure and hard work are cited as causes, but there is usually a mental element of anxiety also. About 10 per cent. of men are smokers, very few (4 per cent.) are drinkers, and syphilis is almost never present (2 cases).

Rheumatism has been put down as a cause, but few patients are really sufferers from articular rheumatism. Many, however, as the disease comes on, suffer from lumbago, sciatica and various forms of muscular pain, which are usually rather premonitions than causes of the disease.

Diabetes was present in three cases, but it is probably a coincidence.

It sometimes follows acute infections and has perhaps a relationship to rheumatoid arthritis, or metabolic and autotoxic rheumatic conditions. The view that it is a "parainfectious" malady, due to the late action of an infection, has been suggested.

Symptoms.—The disease is sometimes ushered in with an acute illness, or an attack of neuralgia. It usually develops slowly with some aching pains in the arm and a slight tremor in the fingers of one hand, oftener the right. For a period of several months the patient perhaps simply knows that he has an arm. Then the tremor becomes distinctly marked in the hand. It gradually extends and involves the leg of the same side, then the other side becomes affected. The neck, face, and tongue are rarely attacked by tremor, and then to a small extent. After or with the tremor there comes on a stiffness in the arms and legs, and indeed of the whole body. With this there is a general contracturing and shortening of all the flexor groups, so that the head and body are bent forward, the fingers are straight but are flexed as a whole on the metacarpus, the forearms flexed on the arm, the trunk is flexed forward on the thighs, and the knees are slightly bent. The attitude gives the idea of extreme senility (Fig. 241). The gait is slow, the steps are short and shuffling; the patient has trouble in starting, stopping, and turning corners, owing to the slowness in initiating new movements in the voluntary muscles. When once started, he may be unable to stop and has to run along. The speech becomes affected in one or two years. The voice is high-pitched, weak, and piping, or senile in quality. There is a slowness in getting out words or in starting a sentence, though after it is begun the words come rapidly. The condition is analogous to the festination in the gait.

Along with the other symptoms, there are often, though not always, sensations of heat, burning, fever, and rarely of coldness. These sensations are felt most in the feet, legs, or arms diffusely. Often there is a general feeling of restlessness and nervousness. Aching pains and a sense of fatigue occur; neuralgic pains are more rare. There are always a peculiar redness and flush in the faces of the patients. Sometimes they sweat profusely. The temperature in the axilla is normal, on the skin it is sometimes increased. The appetite is excellent, often abnormally great, and digestion is good. Visceral complications are rare. Muscular weakness comes on early; it slowly increases, but real muscular paralysis does not occur. The disease ends in rigidity, which makes the patient as helpless as if paralyzed, but the muscles preserve considerable functional power to the last. The deep reflexes are present and not, as a rule, exaggerated; but exaggeration and even clonus occur in a small percentage of cases. As the disease progresses

the tremor increases in extent, and continues with little remission during all the waking hours; later the limbs get more rigid and in a way this rigidity drowns out the tremor; the patient becomes weaker and suffers keenly from sensations of fatigue and exhaustion. At last he can no longer walk; he becomes bedridden and is finally carried off by exhaustion or some intercurrent illness.

It will be seen that the dominant symptoms in paralysis agitans are:

1. Tremor.
2. Rigidity, progressively increasing, with trunk deformities and contractures.
3. Muscular weakness.
4. Sensory, vasomotor and metabolic disturbances.

Further details must be given regarding these symptoms:

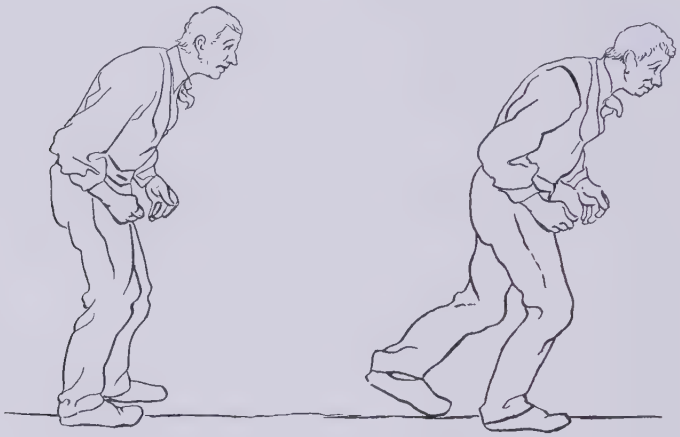


FIG. 242.—Festination in paralysis agitans.

The *tremor* is a coarse one. It ranges from about 6 vibrations per second to 3.7. The average rapidity is 4 or 5 per second, which is about one-half the normal muscular rhythm. But the chief characteristic of the tremor is that it continues when the hand or limb is at rest, while voluntary motion causes it to cease. As the hand rests on the knee it shakes; as it is moved the tremor stops. When held straight out there is no shaking for a moment, but it soon begins. A glass of water is carried safely to the lips. The patient can control the tremor for a moment, especially in the early stages of the disease. These facts about the tremor apply in 90 per cent. of cases, but there are patients whose tremor is slight when the limb is at rest and is increased on voluntary effort. The hands are affected in a characteristic way. The fingers and thumb are slightly flexed and held about in the writing-position; the tremor moves the fingers and thumb as a whole, and they vibrate

so that the one pats the other gently. Sometimes the tremor is one of alternate supination and pronation of the forearm. The neck and face muscles are not usually or extensively involved, the shaking of the head being generally the result of the general bodily tremor. Sometimes one sees a tremor of the lips or neck muscles. The tongue and eye muscles are practically never involved in tremor.

Rigidity.—The rigidity comes on early, and may be the first and even the only prominent symptom. It affects chiefly the flexors of the arms, head and trunk, and legs, producing a characteristic senile position. In rare cases the extensors of the neck are affected and the head is drawn back. Cramps occur, and there is always a sense of stiffness. The muscular movements are slow, especially the initiation of a movement. Once started, a motion may be quickly done. The gait is peculiar; the steps are short and shuffling; the patient may have difficulty in starting; but once started he goes very well; or while walking there may be a sudden running forward. This is called "festination." Rarely there is a tendency to run backward or sideways. Some *muscular weakness* sets in early and slowly progresses, apparently with degenerative changes in the muscles. There is, however, no real nerve paralysis, but rather a muscular inefficiency due to the rigidity and not accompanied with degenerative electrical reactions. The innervation of the facial muscles is weakened and the movements slow so that the face becomes smooth and expressionless and mask-like. The muscles are overirritable to mechanical irritation and the facial responses to blows are exaggerated. The voice in paralysis agitans always becomes affected. It grows weaker, the singing power is lost, then capacity to talk long or loud. The lawyer has to give up trying cases. Speech is slow, difficult and the voice high-pitched, feeble and piping. Examination shows sometimes tremulous movement of the cords, but more characteristic is the slow, weak movement of the cords. In other words, the intrinsic muscles become weak and stiff and a little tremulous like the other muscles of the body.

The handwriting in paralysis agitans soon shows characteristic features. The act of writing becomes difficult; the letters are formed slowly and painfully; they are drawn rather than written. The letters get smaller and more imperfect. Sometimes the writing is distinctly tremulous; more often the characteristic is smallness and lack of finish of the lines, the letters finally becoming obscured. From year to year the words are written more imperfectly and in smaller letters. Tremulousness, slowness, lack of finish of letters and progressive micrographia are the characteristic traits.

The eye movements share the general tendency to functional arrest. The patient stares steadily ahead. Lateral movements both of the eyes

and of head are difficult. But the vision remains good, and with glasses the patient continues to read though convergence is tiring. The sensory nerves do not become involved except that pains of a neuritic or aching and rheumatic character occur. An initial attack of sciatica or brachialgia is not uncommon. The muscular sensibility is exaggerated. This cause a feeling of restlessness that is very annoying. The vasomotor system is lacking in tone and stability. A characteristic flush early shows on the cheeks of the patient. The skin is ruddy, there is a tendency to sweats, and sensations of heat are often noted. The patient likes a cool room. The spinal centres are gradually weakened. Constipation is the rule; sexual power is gradually lost; the bladder after some years becomes irritable and there is a sense of frequent desire to micturate. This, of course, occurs often in the older cases, but it is a vesical not a

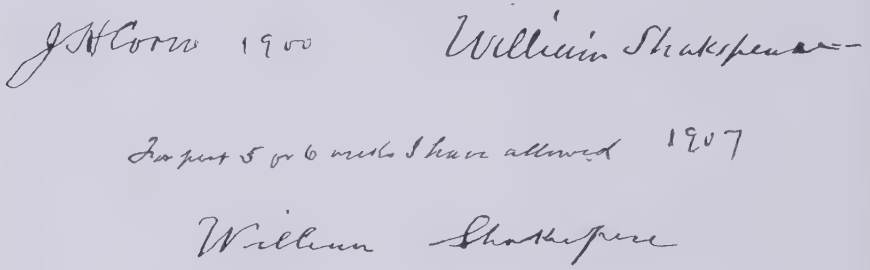


FIG. 243.—Showing progressive micrography. Above, handwriting in 1900; below handwriting seven years later.

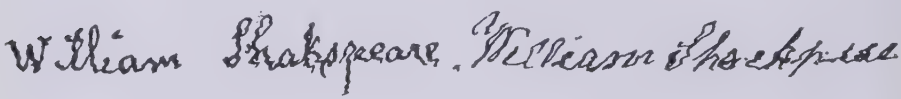


FIG. 244.—Showing tremor in paralysis agitans.

prostatic trouble. Examination shows a congested condition about the neck of the bladder.

The patient is often emotional, but the mind is not seriously affected. The urine is usually about normal, but contains an excess of phosphates. There may be polyuria and less often glycosuria.

Forms.—A not very uncommon type is one with a great deal of tremor and hardly any rigidity, at least not until very late in the disease. About equally common is the type "without tremor" and with rigidity, limitation of movements and deforming attitude alone. Almost always even in these cases a little tremor is sometimes seen. Unusual types of paralysis agitans are the hemiplegic or the monoplegic, and the retrocollic type.

Course and Duration.—The disease slowly but steadily progresses until a full development of symptoms occurs, when it may remain sta-

tionary. It takes about two years for the whole body to be affected, though this varies much. It lasts from three to twelve years or even more. In three cases of mine, death occurred in three, six, and eleven years. Death is due to exhaustion and may be preceded by what seems like a toxic delirium with perhaps some fever.

The **diagnosis** must be made from senile tremor, multiple sclerosis, post-hemiplegic tremor, writers' cramp and wry-neck affecting the ex-

FIG. 245.—Muscular changes in late paralysis agitans.

tensors bilaterally (retrocollic spasm). Senile tremor occurs in the old. It affects the head and there is no rigidity. In multiple sclerosis the tremor is more jerky and is a tremor of intention; there are nystagmus, syllabic speech, and often apoplectiform attacks, eye trouble, and paralyses.

Post-hemiplegic tremor is accompanied by a history of hemiplegia; there are paralysis and exaggerated reflexes and the disease is unilateral. The absence of exaggerated reflexes, the peculiar voice, gait and at-

titude, and the sensations of heat and nervousness often help greatly in the diagnosis.

The **prognosis** is favorable as regards life; unfavorable as regards

FIG. 246.—Cells of the anterior horn of the spinal cord. The right row from a case of paralysis agitans, showing atrophy and pigmentation, the left row from a normal case.

cure, and not very good as to bringing about a cessation of progress in the symptoms. The progress of the malady, however, can be delayed.

Pathological Anatomy and Pathology.—The post-mortem changes are not very marked, and are seen mostly in the spinal cord and brain-

stem. There are congestion and dilatation of vessels in the gray matter, a diffuse increase of interstitial tissue, atrophy and pigmentation of cells. The disease has yet no established pathological anatomy. Degenerative lesions have been found in the anterior cornual cells of the spinal cord, and these resemble somewhat those of senility, but the trouble cannot be explained as just a premature senility of the peripheral neurons, for extreme old age does not present the picture of paralysis agitans. Degenerative changes have been found in the muscles, but these are probably secondary and it is not possible to explain the vasomotor disturbance, the mental disturbances or even the tremor by a muscular lesion alone. The disease seems to impair first the automatically acquired activities, so that to do an accustomed act like writing requires a distinct and persistent voluntary effort. This would lead one to think that the trouble was at the point of co-operation between the voluntary and the automatic mechanism. The tremor and rigidity, without evidence of pyramidal disease, point also to this region and the extrapyramidal tracts. And in old cases at least definite degenerations are found in these tracts.

It is still unknown whether the exciting cause of the functional degeneration is a glandular defect, an infection, or a toxin.

Dr. Thos. W. Hastings has made careful studies of the blood in seven personal cases of mine. In four of them evidences of infection by gonococcus or a micrococcus or both were found.

Treatment.—The most important measure is rest, mental and physical, with plenty of fresh air. No special diet is indicated. Hot baths and mild massage are agreeable and helpful. I know of no climatic cure. The galvanic current produces temporary relief; it should be given daily. Hyoscine hydrobromate, first used by Charcot and introduced into this country by Seguin, is of much value in relieving the tremor (gr. $\frac{1}{100}$ increased). Codeine and morphine are of help if used carefully. A fairly good dose given once in the morning may be tried. Quinine and mineral acids are of much service in relieving the vasomotor and sensory symptoms. Bromide often helps the insomnia and restlessness. Polyvalent vaccines and injections of neo-salvarsan are being tried. Full doses of asafœtida sometimes help. The mind in paralysis agitans is sometimes in an emotional, almost hysterical condition, and patients are easily made better for a time by some psychical influence. Violent exercise does harm. Regular systematic training exercises do some good, vibration is useless and so are hypnotism and osteopathy.

CHAPTER XXVIII

TROPHIC AND VASOMOTOR DISORDERS

Progressive facial hemiatrophy is a disease characterized by a progressive wasting of one side of the face.

Etiology.—It begins oftenest in the young between the ages of ten and twenty. Females are more affected.[1] There is in rare cases a hereditary history. Injury and infectious fevers sometimes start up the trouble. The left side is oftener attacked.

Symptoms.—The disease begins very gradually and shows itself first in patches. The skin gets thinner, there is loss of pigment, hairs

FIG. 247.—Facial hemiatrophy, early stage, showing alopecia and osseous depressions.

fall out, including those of the lids and the areas may have a yellowish appearance. Sometimes the periosteum and bone are affected, and shallow depressions are formed which may be anæsthetic (Fig. 248). The subcutaneous tissue is most involved, the muscles suffer least, and there are no changes in electrical reaction. The muscles of mastication are usually spared. The bone undergoes general atrophy and the lower jaw may be reduced to two-thirds the normal size. The secretion of sebum ceases, but that of sweat may be increased. The temperature falls. There are a sinking in of the eye, narrowing of the lid, and dilata-

[1] About one hundred cases have been reported. Among five, seen by myself, three were in females, two in males. The disease in all cases, so far as could be found, began between the tenth and twentieth years.

tion of the pupil. There is sometimes pain and rarely anæsthesia. The tongue and very rarely the shoulder-girdle or the other side of the face may be involved.

Spasmodic movements of the muscles of mastication have been noted (B. Sachs). Scleroderma sometimes appears on the face or hands. The trouble may be complicated with other diseases of the nervous system, such as epilepsy, myoclonia and psychoses.

The disease progresses rather rapidly at first, but finally comes to a standstill. It does not shorten life.

Fig. 248.—Facial hemiatrophy. (Schoenborn and Krieger.)

Pathology.—There has been found a degenerative neuritis involving the fibres of the trigeminus and its descending root. The weight of evidence is in favor of the trouble being due to disease of the upper cervical sympathetic.

The **diagnosis** is easy. Hemiplegia with atrophy in children, congenital asymmetry, and atrophy from gross lesions of the nerve are distinguished by their stationary character or the presence of severe pain.

Treatment.—There is no treatment known to be of service. Tonics, iodides, and electricity may be tried. Dercum has suggested resecting the trigeminal nerve. It would be worth while investigating surgically the superior cervical ganglion.

Fig. 249.—Facial hemihypertrophy occurring in a giant.

Progressive facial hemihypertrophy is an extremely rare condition only eleven cases having been reported. It is usually congenital in origin, but may develop in connection with giantism, as in a case of my own (Fig. 249).

DISEASES OF THE PITUITARY GLAND

The pituitary gland is composed of an anterior part derived from the buccal ectoderm which has to do with nutritive changes and the development when diseased of acromegaly and giantism; a pars intermedia which produces a substance that stimulates unstriped muscle, produces diuresis and secretes a product which passes into the cerebral ventricles and has some influence on

the growth and functions of the nervous system; a posterior part which consists mainly of neuroglia. Feeding the anterior part to young animals seems to stimulate growth. The posterior lobe extract has no physiological effect. The pineal gland seems to have somewhat analogous functions.

PATHOLOGICAL AND PHYSIOLOGICAL EFFECTS ATTRIBUTED TO THE ACTIVITIES OR DISORDERS OF THE

Pituitary body	Pineal body
Adiposity.	Adiposity (later sometimes a marked atrophy of fatty tissues).
Sexual changes.	Early development of sexual organs and functions.
Genital atrophy and infantilism.	Early bodily and mental maturity (Macrogenitosomia of Pellizi).
Acromegaly and gigantism.	*Physiological action of extracts of the gland.*
Polyuria.	Contradictory reports as to pressor and depressor effect on blood-vessels.
Control of carbohydrate metabolism.	Stimulation of unstriped muscular tissue of intestines, uterus, pupil.
Lowered temperature.	Vasodilatation of genitalia and kidney.
Co-ordinate action with other glands.	Transitory diuresis.
Physiological action of extract of the gland.	Glycosuria.
Pressor and depressor effect on blood-vessels.	Stimulation of metabolism (Berkeley).
Galactagogic effect.	
Stimulation of muscles of pupil, uterus and intestines.	
Modifications of metabolism and bodily growth.	
Modification of carbohydrate metabolism.	

Three disorders are recognized clinically as due to disease of the pituitary gland; giantism, acromegaly, and the Fröhlich syndrome, a condition characterized by stunting of growth, adiposity, and hypoplasia of the genitals (Fig. 250).

Acromegaly is a chronic dystrophy characterized by gradual enlargement of the hands, feet, head, and thorax, and by a dorso-cervical kyphosis.

Etiology.—It affects the two sexes nearly alike. It begins between the ages of eighteen and twenty-six, although a congenital case has been reported. No hereditary influence or definite exciting cause is known. The patients are sometimes naturally endowed with large extremities.

Symptoms.—The disease begins with a gradual enlargement of the bones and soft parts of the hands, feet and head, including the nose, lips and jaws. In women there is amenorrhœa, in men, sexual weakness; slight rheumatic pains, headaches, malaise, mental hebetude, anæmia, and general weakness are present. The patient shows striking contrast between his bulk and his general strength. The skin is dry and there is polyuria.

The hypertrophy affects the soft parts as well as bones. In these latter there are periosteal thickening and hyperplasia, with the result of producing increase in width more than length. The arms are not much involved, nor is the shoulder-girdle, except

the clavicle. The lower jaw is much more involved than the cranium. The tongue, lips and nose are enormously hypertrophied (Fig. 251). The thorax is enlarged antero-posteriorly and flattened. There is characteristic cervico-dorsal kyphosis. There is sometimes dullness over the sternum due to persistence of the thymus. The pelvis may be enlarged, but the hip and leg bones are generally spared. The hands and feet undergo enormous hypertrophy (Fig. 252). The following are some of the measurements in the case that has come under my observation, reported by Adler, and in cases reported by Osborne and Packard:

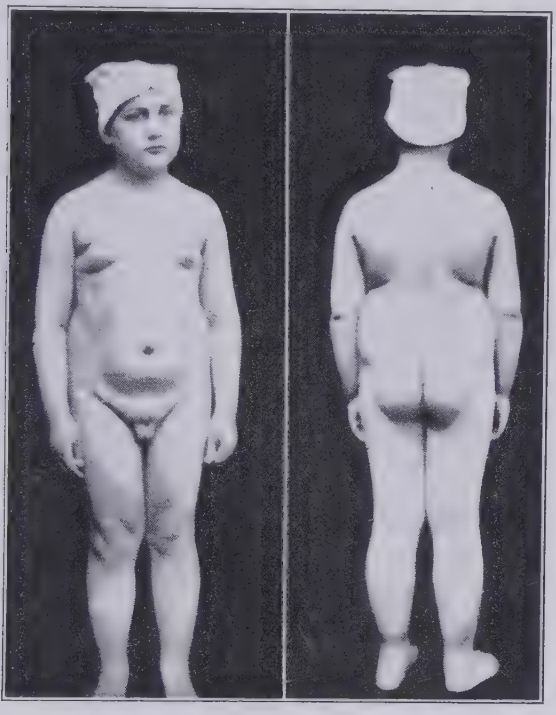


FIG. 250.—Fröhlich syndrome. Tumor of hypophysis in boy of twelve. Adiposity, defective development of genitals; choked discs, headache, nystagmus.

Length of hand	7.6 to 8¾ inches.
Length of foot	11.7 to 12¼ inches.
Cranial circumference	24 to 26½ inches.
Circumference of thorax	44 inches.

The whole body may take part in the growth and some giants have acromegaly, if the disease begins quite early in life.

There may be vasomotor and cardiac disturbances.

Headache, and symptoms of brain tumor may develop owing to the enlarging pituitary body. The patient's mind usually becomes dull and apathetic. Disorders of metabolism occur with bulimia, and glycosuria. The vision is sometimes impaired and there may be temporal hemianopsia due to the pressure of the enlarged pituitary

body on the optic chiasm. Ocular palsies may occur. The muscles may be at first hypertrophied, later atrophied. There are no paralyses and rarely any anæsthesiæ.

The disease runs a very chronic course, lasting ten or twenty years.

Pathology.—There has been found an enlargement of the pituitary body in nearly all cases, and it is probable that the disease is to be regarded as a perversion of nutrition accompanied by hypertrophy and defective function of the pituitary gland.

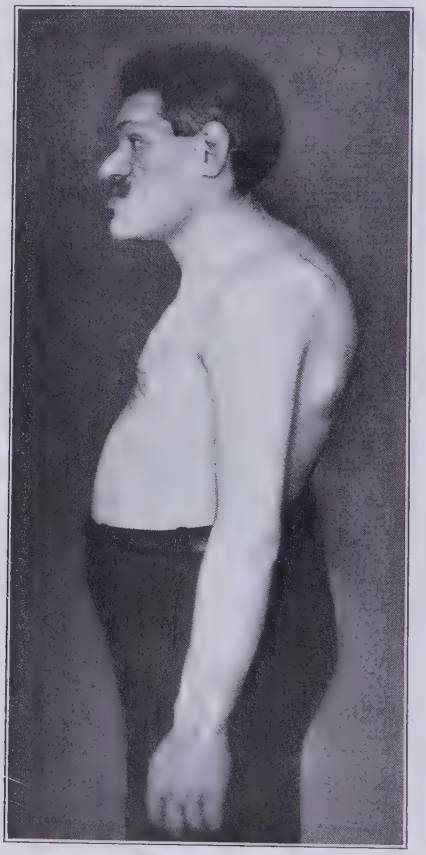


Fig. 251.—Acromegaly.

If pituitary disease begins before the epiphyses of the long bones are united there may be giantism; if it begins later, acromegaly.

The **diagnosis** must be made from congenital enlargements, from so-called giant growth which affects single members, and from osteitis deformans. In the latter disease it is the shafts of the long bones and the cranium, not the face, which are involved.

Prognosis.—Acromegaly is incurable, but it has been arrested, or at least has ceased to progress, and it may not greatly shorten life.

Treatment.—Cases have been reported in which iodide of potassium and arsenic

have arrested the disease. In general, the treatment is only symptomatic, but feeding with pituitary gland has been tried, and it sometimes does good. Removal of a portion of the gland by the surgeon may be required on account of the development of neighborhood symptoms. The use of X-ray and of radium is now being tested.

MYXŒDEMA

Myxœdema is a disease of the thyroid gland, but its symptoms are so largely nervous and mental that a brief description of it is justified here. It is a chronic disorder, due, as a rule, to an interstitial thyroiditis, and characterized by a solid œdema of the subcutaneous tissue, dry skin, loss of hair, subnormal temperature, mental dullness, and even

Fig. 252.—Hand in acromegaly.

insanity and idiocy. It has two forms—the congenital and infantile —causing a condition known as cretinism; and an adult form constituting myxœdema proper.

It occurs most often between the ages of thirty and fifty, and oftener in women (seven to one). It is seen oftenest in temperate climates. Hereditary influence, alcoholism, and syphilis are not predisposing factors; lead poisoning may be a cause.

It begins slowly. The patient is languid and dull, and is unusually sensitive to cold. Voluntary movements are slow; the weight increases and a solid œdema which does not dent on pressure develops in the

face and extremities. The skin gets dry and rough, the hair begins to fall, the temperature is subnormal, 1° to 2°F. Mentally the patient is dull, depressed, and in one-fifth of the cases melancholia, mania, or dementia develop. The muscles are weak, the gait is slow, the voice hoarse and monotonous. There is considerable anæmia and the heart is weak. The pulse is slow and the arterial tension low. Albuminuria is present in 20 per cent. of cases, and hemorrhages may occur.

The pallor, œdema, loss of hair, and mental hebetude give to the face a characteristic expression (Fig. 253). The disease may run a course of six or seven years, the patient dying of cardiac weakness or some intercurrent malady.

FIG. 253.—A case of myxœdema in a man aged forty-four years. (*Murray.*)

The disorder is due in most cases to a chronic interstitial thyroiditis which usually causes atrophy of the gland. It may be produced by artificial removal of the thyroid. The result of this is a defective action of the thyroid, and a consequent poisoning of the system and deposit of mucin in the subcutaneous tissue especially.

The **diagnosis** is based upon the peculiar physiognomy due to the œdema, the loss of hair, subnormal temperature, mental hebetude, and atrophied thyroid.

The **prognosis** is good if treatment is instituted early.

The **treatment** consists in the administration of the thyroid extract in daily doses ranging from five to forty grains or even more. The results are most brilliant, and humanity owes much to Dr. George R. Murray, who first instituted it.

CRETINISM

Cretinism is a form of myxœdema due to atrophy, or defective functioning of the thyroid gland, occurring congenitally or during infantile life. The disease occurs endemically in parts of Europe, but only sporadically and with great rarity in America. Hereditary and family influences are at work in endemic but not in sporadic cretinism. It develops either directly after or in the first three years of life, and shows itself in a stunted growth both of brain and body, most cretins being idiotic dwarfs. The general symptoms are much

Fig. 254.—A cretin dwarf, aged twenty. (*Leszynsky.*)

like those of myxœdema in adults plus the retarded growth of mind and body. The deposits of solid œdema cause peculiar deformities and lead to a characteristic physiognomy. The mind is dull and placid, the muscles are weak, the abdomen is protuberant, the hands and feet are broad and thick; the patients are anæmic, the temperature is subnormal. The arrested bodily growth is such that on reaching adult age the stature may be only twenty-eight to thirty-three inches.

Cretins usually die young, but some survive to the age of thirty and forty.

The treatment is the same as that of myxœdema. Here, too, if the case is seen before adolescence, brilliant results can be obtained.

ANGIO-NEUROTIC ŒDEMA

(*Circumscribed Œdema; Quincke's Disease, Œdema Fugax*)

Angio-neurotic œdema is a disorder characterized by the rather rapid appearance of circumscribed and temporary swellings of the skin and

subcutaneous tissue, and of the mucous membrane. The swellings appear upon different, generally exposed parts of the body. The mucous membranes, especially those of the respiratory and gastro-intestinal tracts may be involved at the same time or independently.

The disease occurs oftenest in early adult life, the average age being from twenty to thirty, but it has been observed in young children and even in the aged. It occurs oftener in females than in males. Hereditary influence plays a part in some cases; and the disease sometimes runs in families. It occurs in neuropathic individuals and families. It is associated with other paroxysmal neuroses such as migraine, urticaria, certain forms of asthma, œdematous purpura and eczema. It occurs oftener in winter and oftener in the early morning hours.

Exhausting occupations predispose to it. The exciting causes are sudden exposure to cold, slight traumatisms, fright, anxiety, grief, and the ingestion of certain kinds of food, such as apples, strawberries, or fish. A peculiar form of this œdema seems to develop in connection with menstruation.

Symptoms.—The disease appears without much if any warning. In a few minutes or hours there develops a circumscribed swelling upon the face or arms or hands. This swelling varies in diameter from one-half inch to two or three inches. It may be dark reddish or rosy or it may be pale and waxy. It does not easily pit on pressure. There is sometimes a rise, sometimes a fall in local temperature. It is accompanied by sensations of tension and stiffness, scalding, burning, and sometimes itching, but there is no actual pain. The swelling is usually single, but it may be multiple. It is located most often upon the face, next upon the extremities, particularly the hands; next on the body, then in the larynx and throat, and then on the genitals. The swellings last from a few hours to two or three days. Between the attacks the patient feels well. They are apt to return at intervals of three or four weeks to several months. Sometimes they are brought out only by certain peculiar exciting causes, such as indigestion or mental anxieties or emotional disturbances. When the disease attacks the larynx or throat, serious symptoms of dyspnœa and suffocation may appear; surgical interference may even be called for, and death has been known to result. The œdema may attack the stomach, producing nausea, vomiting, and great gastro-intestinal distress, or the intestines causing colic and diarrhœa.

The **pathology** of this neurosis is imperfectly known but is somewhat as follows:

The disease is associated with and is allied to a group of neuroses or angio-neuroses including migraine, urticaria, certain forms of asthma and purpura.

The victim possesses a constitutional defect which is sometimes inherited and transmitted. This defect may be a dysthyroidism, or other disorder of the endocrine cells. It shows itself in weakness in the chemistry and cellular functions of the gastro-intestinal tract, allowing entrance into the blood of heterogeneous proteins (anaphylaxis).

The incident of an attack of œdema (anaphylactic shock) is brought about by depressing emotions, trauma, shock, exposure, certain foods ingested.

Then the heterogeneous proteid in the system attacks the vasomotor nerves at certain points, because there is an irritant or extra-point of weakness at these points. There follows an excessive permeability of the vascular endothelium, and transudate of serum.

Diagnosis.—The symptoms of the typical disease are so peculiar that they are easily recognized. The spontaneous appearance of the œdema, its recurrence at certain intervals, and the absence of serious pain and evidences of inflammation are sufficient usually to enable us to recognize it. Giant urticaria is a disease resembling neurotic œdema and probably closely allied to it. The blue and the white œdema of hysterics only exists with other stigmata of hysteria.

The **prognosis,** so far as cure is concerned, is not very good. The attacks, however, can be ameliorated, and the disease itself is not serious, as regards life except when it attacks the larynx. Most of the patients whom I have seen have eventually recovered.

The **treatment** consists in the adoption of such measures as will give tone and stability to the nervous system, and the gastro-intestinal tract. In some cases great attention must be paid to diet. Internally, mineral acids and strychnine may be of some value. A systematic and prolonged use of arsenic has been followed by cure; urotropin has been used with success, and so also has nitroglycerin, and horse-serum (in a case associated with purpura). The lactate of calcium ʒi daily is sometimes helpful. Gastro-intestinal crises may require morphine. In laryngeal œdema, intubation should be promptly done.

CHAPTER XXIX

THE DISORDERS OF SLEEP

INSOMNIA, HYPNOTISM, MORBID SOMNOLENCE, CATALEPSY, TRANCE, LETHARGY, THE SLEEPING SICKNESS

The most conspicuous phenomenon of sleep is the loss or partial loss of consciousness. With this the regulating mechanism of the mind is in abeyance. Mental processes go on, when at all, by free associations or in accordance with the emotional trend or as activated by sensory and somatic irritants.

The cause of sleep is not exactly known. We know that sleep is a normal resting phase of brain action, a kind of cerebral diastole. It is probable that the brain neurons by their activity throw off products of metabolism which tend to benumb and inhibit their waking physiological functioning. It has been found that the brain is slightly anæmic during sleep, but this is part of the process not the cause of it. Other organs of the body rest somewhat also during sleep, and especially is this true of the muscular system. There are slight changes in pulse, respiration, blood-pressure and other bodily functions in sleep but these are described adequately in physiological text-books.

Normal sleep varies much in accordance with age, sex, the individual, and, to a slight extent, with occupation, race and climate. The infant sleeps at first nearly all the time, later fourteen to sixteen hours, but its sleep is light. Young children sleep ten to twelve hours. The adult needs about eight hours, while the aged live healthfully with but six. Women need half an hour or an hour more than men. A few persons, generally men, need nine, ten, or even twelve hours' sleep daily; others require only six. Brain workers, as a class, take less sleep than laborers. Sleep is sounder and longer in cold climates and among northern races.

The disorders and perversions of sleep include insomnia, morbid somnolence, morbid dreaming, sleep-walking, hypnotic and trance states. They are disarrangements of a normal function, brought about by various causes. Sleep itself may be the morbid expression of a disease, as in narcolepsy, the sleeping sickness of Africa, and the somnolence of the senile brain. Conditions allied to normal sleep exist in the hypnotic and trance states. Among all these conditions, practically, insomnia is the sleep disorder which most annoys humanity, and it comes next to food and raiment and human love in the concern of civilized man.

INSOMNIA

Insomnia is a condition in which persons suffer from insufficient and disturbed or unrestful sleep. The term "insomnia" covers all these deficiencies, but the old Roman writers used the word *exsomnia* for a condition of excited wakefulness, such as came with morbid mental exaltation and the delirium of the Bacchic priestess.

Insomnia is a symptom of many disordered physical and mental conditions, and it has been considered elsewhere in connection with the most important of these. It occurs in its most serious and persistent forms in painful diseases, in the psycho-neuroses, and in manic and depressed states. Here one sometimes finds cases of apparent entire absence of sleep for prolonged periods. This *ahypnosis* is not, however, absolute. Man can exist without sleep for about the same time that he can go without food, *viz.*, 3 or 4 weeks, but he cannot live without it.

Etiology.—Insomnia of mild grades occurs often apart from any very serious physical disturbance. It may be a hereditary or family trait not to sleep easily or well. More often it is acquired by bad habits of life, by disturbing environments, by anxiety of mind and by excessive use of drugs, tobacco, tea and coffee. It may be the result of endogenous irritants, such develop in cases of poor digestion, in faulty metabolism or in hyperthyroidism.

Disease of the heart and arteries may lead to insomnia, and under this head come the cases which occur in Bright's disease with sclerotic arteries and anæmic brains. Disorders of the stomach lead to disturbed sleep oftener than to complete insomnia, and the liver, when inactive, causes somnolence rather than the contrary. Insomnia is a disorder especially of the active periods of life, though children suffer from it also. In their case it is usually due to some definite physical cause, such as pain, indigestion, emotional excitement; yet one not infrequently meets children who from early life go to sleep with difficulty. It is in this period of life that the active and bizarre disorders of sleep occur, such as sleep-walking, night-mare and *pavor nocturnus*. The aged have insomnia but do not suffer so much from it. Women have insomnia rather less than men; and brain workers more than muscle workers. Insomnia is more common in cold climates, largely because there is more brain work done there. High altitudes tend to cause insomnia, at least, till the system becomes accustomed to the change.

Anxiety of mind and brain fatigue lead to insomnia; and very often to the morning type. The patient who carries a worry wakes at 3 or 4 A.M. instead of his usual hour. Insomnia is a warning symptom of the approach of neurasthenia and of certain mental disorders, especially melancholia and the obsessive psychoses.

Summary: Insomnia is due to

1. Hereditary and constitutional conditions.
2. Habits of life: of work, of eating, of environment and of too violent exercise.
3. Irritative conditions such as pain, pruritus, indigestion.
4. Toxic agents, tobacco, tea, coffee, cocaine, morphine.
5. Endogenous toxins, gastro-intestinal, glandular.
6. Vascular degenerative disease of the brain.
7. Psychic causes; fatigue, worry, the onset of psychoses.

Treatment.—The enumeration of the causes of insomnia, covers a large part of the discussion on treatment. For treatment involves a consideration of the cause, then the laying down of rules of hygiene, the arrangement of daily work and play, the times of eating, diet, exercise, the hours of retiring and the amount of sleep needed. Nearly all these points have been discussed under special chapters. I shall discuss now only certain particular medicines and measures used for the symptom insomnia.

The older physicians in treating sleeplessness, used to depend largely on hyoscyamus, camphor, opium, and the fetid drugs, such as asafœtida, musk, and valerian. Hyoscyamus is still used. The hydrobromate of hyoscine, in doses of gr. $\frac{1}{100}$ to gr. $\frac{1}{50}$ or more, is the best form. Hyoscine is indicated in the insomnia of delirium and mania which is accompanied by motor activity. Chloral hydrate still holds its own as one of the surest of hypnotics. The dangers involved in its use have been somewhat exaggerated, though they are sufficiently real. Doses of gr. x. and gr. xv. are often quite large enough, but in alcoholic insomnia it may be given in twice the above amounts, guarded with ammonia and digitalis.

Bromide is one of the best hypnotics but it has to be skilfully used. The immediate effect is simply sedative, and sleep is not produced unless large doses are given. Some persons are even kept awake by average doses (gr. xv. to xx.). In insomnia, therefore, bromides are best prescribed in doses of gr. xxx. twice in the evening for not over four days. By the third evening sleep is generally secured. A bromide habit is rarely formed, and is in itself not seriously injurious. The bromides alone are hardly strong enough hypnotics for the insomnia of delirium and mania and they are not so efficient in the weak and aged. Paraldehyde ranks close to chloral in its value as a hypnotic. In some persons it disturbs the stomach, but not in all, and it may be used as a hypnotic for months without its power being impaired. It is a disagreeable drug, and there is nothing so far as I know, that palliates its offensiveness. I prefer, however, to prescribe it in ʒi. doses poured upon a teaspoonful of pow-

dered sugar. The paralydehyde habit may be formed and it is bad like all drug habits, but less serious than chloral or opium addiction.

Amylene hydrate is a hypnotic of properties similar to those of paraldehyde, but less disagreeable. It is given in doses of about one drachm. Lupulin in large doses, gr. x. to gr. xx. is a fairly good hypnotic. Chloralamide is more agreeable than chloral, though it acts practically in the same way. Sulphonal, trional, veronal, medinal and adalin, all act in much the same way; but vary in strength and quickness of absorption and elimination.

Among the antispasmodics are several drugs which occasionally answer well in the insomnia due to nervous irritability. For example, tincture of valerian and spirits of lavender are sometimes adequate as sleep inducers. Ten grains of aspirin act sometimes as a harmless hypnotic. A bottle of beer, or a drink of whiskey are hypnotics but are dangerous medicines, for their effects wear off and larger doses are soon demanded. Safer measures are the taking of digestive food just before retiring or on too early waking. A glass of hot milk or hot malted milk often works successfully.

Besides drugs, there are many hygienic or mechanical measures to which the physician may successfully resort—listening to monotonous noises, reading dull or heavy books, counting, or keeping before the fancy some blank or wearying picture—

> "A flock of sheep that leisurely pass by
> One after one; the sound of rain and bees
> Murmuring; the fall of rivers, winds and seas,
> Smooth fields, white sheets of water, and pure skies."

In cases of purely bad habit insomnia, psycho-therapeutics even to the point of actual hypnosis may be tried.

Mechanical remedies have nearly all for their purpose the withdrawal of the blood from the brain to the skin and abdominal viscera. Hot footbaths or warm general baths, cold douches down the spine, abdominal compresses, beating the limbs with rubber hammers, massage, all are at times efficient hypnotics. Persons who suffer from insomnia should sleep in cold rooms, the head should not be too high or very low, and in most cases they are better without late suppers, even though these be light. Mental work should be laid aside several hours before retiring and the evening devoted to quiet conversation and reading or amusements that do not actively excite the nerves. Many persons live in good health though they sleep in the day and stay awake at night. Despite this, it is true that the best time for sleep is at night, and that the old maxim, "Early to bed," is a sound one. Yet it is not the early bird that gets the worm so much as the bird that has slept enough. The human system

requires a certain amount of sleep and should have it. The industrious and ambitious often try to train themselves to shorter hours, but though they may succeed for a time, nature will not be cheated out of her due and health suffers in the end. It is a widespread custom in some countries to take a short nap in the daytime, and the custom is a good one, for the weak and aged and over-worked.

In early life, at least, a habit of going to sleep when desired can be formed by careful attention and exercise of the will. It is a most useful asset throughout life.

PERVERSIONS AND DISTURBANCES OF SLEEP

Sleep is said to reach its deepest stage in from one to two hours after it begins. There is then after this a gradual lessening of the depth of sleep. Probably there are great variations in this rule, for many persons seem in soundest slumber several hours after falling asleep. But, at any rate, there are lighter stages of sleep at its inception and toward its end. These are the favorite times for dreams, and at this period also there develop the peculiar phenomena of sleep-drunkenness.

Dreams.—When sleep is perfect and profound, dreams afterward remembered do not occur. Dreaming is, therefore, a morbid symptom, although often of trivial significance, especially if it occurs at about the time of natural waking, when slumber is, in its physiological course, passing into the lighter stages. In sleep, no matter how light, the action of the regulating centre, which directs thought, controls emotion, and exhibits itself in volition, is suspended; the physical mechanism, if excited to action at all, works without purpose, like a rudderless ship at sea. Ideas and emotions succeed each other by the laws of association, but are not properly correlated, and judgment and logical reasoning are gone. As a rule, dreams are made up of somewhat ordinary ideas and fancies incoherently associated, and shifting too rapidly to call up much feeling. When from some point in the body painful sensory excitations do produce disagreeable images, emotions of a most violent kind may be felt.

The Freudian interpretation of the dream as expressing symbolically or otherwise a wish is one that does not concern the clinician unless he believes in the value of psycho-analysis.

Nightmare is a disorder nearly always symptomatic of an irritation in some part of the body. The usual causes of it are some digestive disturbance (repletion) and cardiac disease. Persons of a nervous temperament are more subject to it; and there are individuals who suffer from it all their lives. The popular belief that sleeping on the back favors it is, in general, a correct one. When nightmare occurs in cardiac disease

a certain position, semirecumbent or on the right side, must be maintained, or the painful fancies will awaken the patient. Healthy people can get sound sleep whether lying upon the back, the side, or the stomach; but light sleepers, and those with sensitive abdominal viscera, generally find that the position on the right side is the most comfortable and less provocative of unpleasant dreams. Prolonged mental or physical strain, excitement, and worry, predispose to nightmare. A high proteid diet, excess of sweets in diet, excessive use of strong liquors, coffee and tobacco, have a similar tendency.

Pavor nocturnus, or night-terrors, is a sleep disorder peculiar to children. It is allied to nightmare on the one hand and sleep-drunkenness on the other. It differs from the former condition in that the child continues to suffer from the distressing fancies for some time after he is awake. Night-terrors occur usually one or two hours after sleep has begun. The child wakes up screaming with fright, and perhaps runs about the room or seeks its parents for protection against some imagined harm. The disorder occurs in weakly, anæmic, nervous, or rheumatic children. It is due sometimes to what has been termed lithæmia or, as the older writers put it, rheumatism or gout of the brain. Digestive disturbances, worms, dentition, hereditary syphilis, mental strain, fright, and excitement are placed among the causes. It sometimes appears to be a paroxymal neurosis allied to epilepsy and some cases are really epileptic. The disorder is usually harmless and the prognosis favorable.

Somnolentia, or sleep-drunkenness, is a condition of incomplete sleep in which a part of the faculties is abnormally excited while the other is buried in repose. It is a kind of acted nightmare or somnambulic delirium. The person affected, is incoherent, excited, and often violent. He experiences the delusion of some impending danger, and while under it acts of violence have been committed. A patient of mine was in the habit of waking and attacking his beloved wife. The condition is one of medico-legal importance, therefore, and has been discussed by writers on that science (Wharton and Stillé). Minor degrees of it are often noticed in children and in adults who are roused from a very profound sleep. It at times becomes a habit and a most annoying or dangerous one. The disorder in its severe form is fortunately very rare.

The treatment of morbid dreams, nightmare, and pavor nocturnus must be directed to a removal of the causes. Tonics, cardiac stimulants, laxatives, antirheumatics, attention to diet and time of eating are especially important. Change in surroundings is often necessary. Among symptomatic remedies the bromides are the best, combined sometimes with alkalies and salicylates. In somnolentia the patient should be prevented from getting into too profound sleep. He may be

awakened once or twice during the night, or take a nap in the daytime. The head in sleeping should be raised and the body not too heavily covered.

Somnambulism.—Somnambulism is a condition similar to hypnotism or the mesmeric state. In it volition is abolished and the mind acts automatically under the dominance of some single idea. It is an acted dream. Sight, hearing, and nearly all the avenues of sense are closed. The sleepwalker avoids obstacles and performs ordinary acts automatically like an absent-minded man, which in reality he is. All those mechanisms which have been trained by constant repetition to act automatically like that which preserves equilibrium are active, and their powers may even be heightened, so that the somnambulist may walk along roofs or on dangerous roads and thread intricate passages without harm. The automatism of the somnambulist may continue for hours, until a journey has been performed or a task completed. He may carry out with success familiar mathematical calculations, write a letter, or work upon a picture, but he only follows along the lines established by constant iteration in his waking moments. He can originate nothing new. He is roused from his state with difficulty, and when out of it he remembers nothing of what has occurred.

Somnambulism occurs oftener in neuropathic persons and it may be hereditary. Its exciting cause is often over-eating, or mental strain and excitement. Sleeping with the head too low is another cause. The habit being once established, however, attacks occur without apparent cause. The disorder occurs oftenest in young people about the age of puberty, and it then attacks the sexes alike. Later in life women are more often affected. The disease is fostered sometimes at school by the attentions of the schoolmates. The attacks are likely, after a time, to become periodical, occurring every week, fortnight, or month. The somnambulic state may come upon a person in the daytime. It is then regarded as spontaneous trance, or hypnotism. It is not the case, however, that persons who are easily hypnotized are usually somnambulists, though the reverse may be true. Somnambulism is a term that should include not only sleep walking, but sleep talking.

In the treatment of somnambulism the patient's surroundings must be investigated, and unfavorable influences, such as excessive fatigue and excitement and such as may occur at school or from injudicious nurses, be removed. He should be prevented from sleeping too soundly, the head should be raised, the clothing light, the diet regulated. Remedies like iron, quinine, phosphorus, and cod-liver oil may be given. When the patient is discovered in the somnambulistic state he should not be awakened, or at least not until he is safely back in bed.

HYPNOTISM, TRANCE, MESMERISM

Major hypnotism is a morbid mental state artificially produced and characterized by (1) perversion or suspension of normal consciousness; (2) abeyance of volition; (3) a condition of suggestibility leading the patient to yield readily to commands or external sense impressions; and (4) intense narrowing and concentration of the attention upon some idea or feeling suggested by the hypnotizer.

Minor hypnotism is a state closely bordering on normal sleep in which there is a dulling of consciousness and a condition of suggestibility and narrowing of the attention field.[1]

The proportion of persons of all ages found by Beannis to be hypnotizable was about eighteen or twenty per hundred. Children up to the age of fourteen are very susceptible. After the age of fifty-five susceptibility lessens. Men are almost as easily affected as women; but persons of a docile mind and those trained to some degree of mental discipline and capacity for submission, such as soldiers and artisans, are more sensitive. In this country the percentage of hypnotizable subjects is less than it is in Europe. Hysterical and insane persons are not very susceptible. Those who have been mesmerized once are more easily affected afterward, and may even pass into the state involuntarily.

Methods.—There are two ways of inducing hypnotism: the fixation method and the suggestive method. The former and older plan, devised by Braid, is to make the patient fix his eyes for five to ten minutes on some bright object at a distance of six or eight inches from the eyes and a little above the horizontal plane of vision. Practically, it is sometimes the custom among practising hypnotizers to give the patient a dose of paraldehyde.

In the "suggestive method" devised by Liébault and Bernheim the subject is placed in a chair in front of the operator. The operator then talks to the subject in a firm and confident voice, assuring him that he will go to sleep in a short time; telling him to make no resistance, that his sleeping will be natural, that nothing will be done to worry or fatigue him, that he will dream pleasant dreams, that he will wake up feeling better; then that he is feeling drowsy, his eyes are heavy, objects look confused, the lids are falling, they are closed—in a moment more the patient goes off to sleep. This requires some little time—five to fifteen minutes. It may fail the first time and succeed the second.

Hypnotic states can be self-induced by vigorously fixing the attention upon some object. The ecstatic states of the saints are forms of

[1]The definitions of hypnotism vary with the psychologists and the time of year. The above is only a descriptive definition with no reference to dissociations of personality or the thresholds of consciousness. I may add that the word hypnotism is used in two senses. It indicates a mental state and a general topic.

hypnotism; so also are the trance states in which some clairvoyants and spiritualistic preachers place themselves; this same curious phenomenon is at the bottom of the "mind-healing" sciences, and it enters into rational therapeutics and orthodox religion. The capacity of the human mind for hypnotism or semihypnotic states is, therefore, a most curious and important fact.

Symptoms.—The person who has been hypnotized at first sits or lies quietly in the position he has assumed during the manipulations of the operator. No notable physiological changes occur, as, for example, in the pulse, respiration, temperature, pupils, skin, etc. Some increase in the cerebral blood-supply, however, is said to be present. The patient will now respond automatically to any outside command or will be dominated by any idea which is suggested to him. He will talk, or walk, or run, or gesticulate, assume expressions of fright, anger, or joy, entirely in accordance with the command given. Apart from these commands, he is entirely dead to the outside world. He hears, sees, smells, tastes, and feels nothing. He can be burned, cut, or injured without showing any signs of feeling. At a suggestion he may be made cataleptic, somnambulic, or paralytic. This state is termed *somnambulistic trance.* If left to himself, he gradually sinks into a deep sleep, from which he can with difficulty be roused. After a time, rarely more than one or two hours, he awakes as from ordinary slumber. This latter state is called trance coma, or *lethargic hypnotism.*

The phenomena of hypnotism depend upon the wonderful sensitiveness and quickness of the subject in responding involuntarily, with all his nervous energy, to outside suggestion. Dishonest persons may learn the latter trick and thus simulate the hypnotic state. Traveling mesmerizers utilize such persons largely; hence no confidence can be placed in the phenomena exhibited by them.

Minor hypnotism is produced by the "suggestive method" of hypnotizing. By this latter plan patients are thrown into various degrees of the hypnotic state from slight drowsiness to lethargy, but they are not somnambulic, and do not become cataleptic or anæsthetic.

Patients naturally come out of the mesmeric state through the channel of deep sleep or lethargy. Ordinarily they are dehypnotized by word of command, or by a pass of the hand, or any impression which the patient expects to be used for the purpose.

Hypnotized persons have been observed to have a diminution in the spinal reflexes and a muscular hyperexcitability. They sometimes show a most extraordinary exaltation of visual, auditory, or other special sense.

Therapeutics.—The practice of using major hypnotization is usually injurious, if repeated much, tending to exhaust the nervous force and weaken the will. It should be done only with the greatest care. Its

utility in therapeutics I greatly doubt. It may relieve symptoms in the hysterical for a time, but it cannot be of permanent benefit and is likely to lead to actual harm.

The induction of minor hypnotic states by suggestion is not harmful if carefully and moderately employed. Its practical results, however, are not great. It has its value in pedagogy, among children, in defectives, obsessives, and in morbid habits. The general popularization of hypnotism by means of mind cures, Christian science, etc., accomplishes its results at the expense of mental demoralization, and faith-healing institutes may be pernicious elements in society.

MORBID DROWSINESS AND SOMNOLENCE

This is a common symptom, which may be due to one or more of he following causes: 1. Old age when there is a weakened heart and cerebral arterial sclerosis. 2. Cerebral syphilis. 3. Endogenous toxæmias such as result from constipation, gastric repletion, atonic intestines, and the metabolic disturbances in obesity. 4. Concussion of the brain. 5. Disorder of the pituitary or thyroid glands. 6. Climatic conditions such as extreme cold. 7. Constitutional defects.

A very common cause of drowsiness is dyspepsia attended by some torpidity of the liver, the condition popularly known as "biliousness." Syphilis is more likely to cause insomnia, but in its parenchymatous form somnolent conditions may be produced which are of serious significance. Drowsiness occurs from the effects of severe cold. It sometimes develops when persons change their surroundings, especially on going to the seashore, for low levels and a high degree of atmospheric pressure seem to promote sleep. The drowsy state that sometimes follows concussion of the brain is a familiar phenomenon. Some persons, no doubt, acquire the habit of drowsiness. At first the trouble may have been induced by indigestion, "biliousness," or malarial infection, but it persists after the cause is removed. Such persons can hardly sit through a lecture, a church service, or any exercise requiring quiet and attention. Morbid somnolence is sometimes produced by disordered activity of the pituitary and thyroid glands. The patients are unable to keep awake except when actively engaged in work. Sitting at a table or in the chair they fall asleep and sleep so profoundly that they fall to the floor.

Morbidly Deep Sleep.—Certain persons, when they sleep, pass into an almost lethargic slumber. Persons who suffer in this way often sleep a longer time than normal. They are awakened with difficulty and then suffer with headache or disagreeable sensations throughout the day. Instances in which persons retire at the usual hour, but can with great difficulty be roused in time for the ordinary duties of the day, are not

rare. Some of these are illustrations of the vice of indolence, but in other cases there is an absolute need of nine, ten, or even fourteen hours of sleep.

This disorder of sleep is most liable to occur in the young and in those of nervous temperament. It often seems to be a constitutional condition, for which nothing can be done. In other cases it results from over-feeding and indolent habits.

Paroxysmal Sleep, Narcolepsy, Sleep Epilepsy.—It sometimes happens that persons suffer from sudden attacks of unconquerable drowsiness; they fall off into slumber despite every effort of the will. These are more than drowsy sensations, for sleep, or a state resembling it, cannot be kept off. Some of these cases are of a purely nervous character, *i.e.*, the trouble is not due to a toxæmia or to organic disease, but to a paroxysmal change in the nervous centres of a vascular or chemical character, causing sleep. It may be that the patient is epileptic and sleep seizure takes the place of the ordinary epileptic spasms.

Fig. 255.

Cases of epileptic sleep, or narcolepsy, and allied forms are not of frequent occurrence. Females are rather more often affected than males, and the susceptible age is from fifteen to forty. The disorder is brought on sometimes by fright, over-strain, and humoral poisons acting on a predisposed nervous system.

The course is chronic and relief is not always obtained. It should be remembered that syphilis, malaria, or anæmia, and indigestion may be elements in the trouble which are important, if not fundamental. Bromides in small doses are often useful factors in treatment. Change of occupation, of mode of life, or of climate may be essential to a cure.

Catalepsy, Trance.—Most of the so-called cases of prolonged sleep, lasting for days or weeks, are cases of spontaneously developed mesmeric sleep in hysterical women, or cases of incipient insanity (dementia

præcox or stuporous melancholia). The phenomena in these cases may take the form of *catalepsy*, with waxy rigidity of the limbs, or of a trance state. In cataleptic states the limbs may be placed in various positions and will remain there for several minutes (Fig. 255). In lethargy or trance states the patient may be plunged into a deep and prolonged unconsciousness, lasting from one day to several years. These are the "sleeping girls" of the newspapers. Others are persons of a too ready susceptibility to mesmeric suggestion, who get into a morbid habit of going into mesmeric sleep spontaneously.

The duration of the attacks of trance lethargy is from a few hours to ten years. Ordinarily, however, profound trance sleep lasts not more than a few days, while those cases in which the sleep is from mesmeric suggestion lasts but a few hours.

The katatonic patients after a few weeks or months gradually awake, become excited, and then pass into a condition of dementia or into catalepsy again.

Morbid Sleep from Organic Disease.—Prolonged and excessive sleep occurs as the result of syphilis of the brain, brain tumors, and the degenerative changes in old age and insanity. It has been noted especially in tumors of the basal ganglia and third ventricle.

Organic diseases of the brain tend to produce conditions of mental weakness, hebetude, or comatose states, rather than anything allied to sleep.

Accidents of Sleep.—Owing to the fact that sleep is a resting state of the organism, and that many of its functions are lowered, or their cerebral control lessened, peculiar crises, or physiological and pathological disturbances of nervous equilibrium, occur. Attacks of gout, of asthma, and of pulmonary hemorrhage are most liable to occur during the early morning hours. Deaths and suicides occur oftener in the forenoon, but births oftener at night. Epileptic and eclamptic attacks occur with much frequency at night. Involuntary emissions of spermatic fluid, or gastric crises, and incontinence of urine are among the pathological incidents of sleep.

Prædormitial Shocks.—Sudden attacks of starting of the whole body, shock-like in character, accompanied by peculiar feeling in the head or occiput, not infrequently attack persons as they are dropping off to sleep. They indicate fatigue and are of slight significance.

THE SLEEPING SICKNESS OF AFRICA

The sleeping sickness of Africa is a disease, caused by a species of flagellated protozoa, belonging to the genus Trypanosoma. This parasite multiplies in the blood, and causes a slowly developing malady char-

acterized by somnolence, mental apathy, paralysis, and finally death. The Trypanosome which causes this sleeping sickness is the *T. Gambiense*, first described by Dr. J. E. Dutton.

Etiology.—The disease prevails mostly along the west coast of Africa, but it has extended, thence, to other parts of that continent, particularly equatorial Africa. It was formerly confined to the negroes, but recently Europeans have suffered from it. It affects particularly natives who live upon the shore of the rivers and lakes. The Trypanosome is a worm-like parasite which is found among the red blood-cells (Fig. 256). They are not very numerous, and several microscopical examinations may be needed to find them. In late stages of the disease, the parasite is found in the cerebrospinal fluid, while early in the stage it is found in the lymphatic glands and channels. The common way of the propaga-

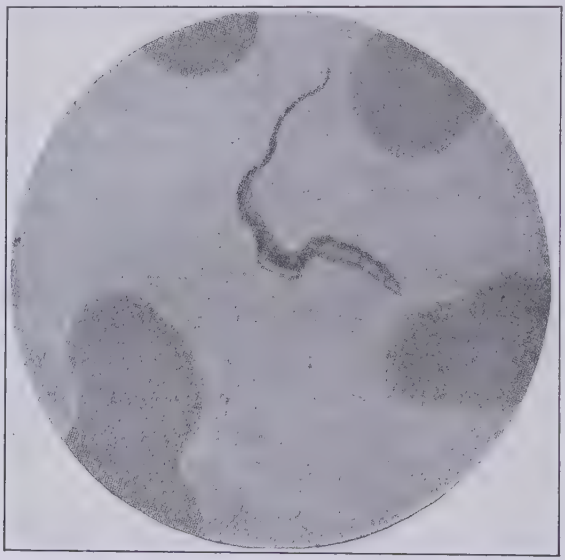


Fig. 256.—The blood in sleeping sickness, showing spirillum.

tion of the Trypanosome is through the tsetse-fly, and it is conveyed back to man by the bites of these flies.

Symptoms.—It is stated by Dr. David Bruce that after the Trypanosome has infected the body, it may be from three months to three years before it gets into the cerebrospinal fluid, and begins to produce definite symptoms. During this stage of incubation, there is some enlargement of the lymphatic glands, and sometimes a fever of irregular type. When the disease begins to affect the patient, he becomes apathetic and disinclined to exert himself. Headache and indefinite pains are complained of. The pulse is rapid and weak. The cervical and other lymphatic

glands are enlarged, the gait is unsteady and shuffling, the muscular powers diminished, the voice is weak, and tremors of the tongue are noted. The symptoms increase until after weeks or months the patient is confined to his bed, lying in a somnolent condition. The patient then begins to emaciate, becomes semicomatose, and in two or three weeks the temperature falls and death occurs.

Pathology. -Dr. F. V. Mott has shown that there is a characteristic appearance in sections of the brain, which is found in no other disease. This is a condition of meningo-encephalo myelitis, which affects especially the medulla and the base of the brain. The inflammation can be traced along the blood-vessels into the substance of the nervous system, the perivascular lymphatics being crowded with lymphocytes. Modern trypanosomiasis is, however, according to Bruce, a disease of the lymphatic system, though its characteristic symptoms are shown upon the nerves and muscular system.

Diagnosis.—The diagnosis is difficult in the early stage of the disease. Later, the examination of the blood and lymphatics, and the peculiar progressive weakness of the body, tremor of the muscles, unsteady gait and somnolence are characteristic.

Prognosis.—Every case of the disease dies sooner or later, according to Dr. Bruce.

Treatment.—Up to the present time the most effective drug has been some preparation of arsenic. The prophylactic measure for the sleeping sickness is to kill off the tsetse-fly.

CHAPTER XXX

CRANIO-CEREBRAL TOPOGRAPHY

The object of cranio-cerebral topography is to map out upon the scalp the underlying fissures, convolutions, and other parts of the brain. Practically, the principal points to be determined are the position of the longitudinal, Rolandic, Sylvian, and parieto-occipital fissures and the lower outline of the brain.

The measurements are based chiefly upon the known relations of certain landmarks on the skull to the parts beneath. These landmarks are the glabella, bregma, lambda, stephanion, asterion, and pterion, which are points at the junction of the various sutures with each other and with certain ridges or protuberances. Their position is shown in the cut (Fig. 252) except that of the glabella or prominence just above the nasofrontal suture. The inion is identical with the occipital protuberance.

The following rules are based upon the observations of Heftler, Thane, Reid, Horsley, Fraser, and myself:

1. The longitudinal fissure. This corresponds with the naso-occipital arc.

II. The fissure of Rolando. Measure the distance from the glabella to the inion; find 55.7 per cent. of this distance, and the figures obtained will indicate the distance of the upper end of the fissure of Rolando from the glabella. It should be about 48 mm. behind the bregma in male adults, 45 mm. in women, 30 to 42 mm. in infants and young children, respectively.

The fissure runs downward and forward for a distance of about 10 cm. measured on the scalp, the real length being about 8.5 cm. The fissure makes an angle of about 67° with the anterior part of the longitudinal fissure. The lower third of it is more vertical, and the lower end is 25 to 30 mm. behind the coronal suture. The fissure is shorter in children.

III. The fissure of Sylvius runs nearly horizontally, and lies either under or a little above the uppermost part of the parieto-squamous suture. *This suture*, the *external orbital process*, and the *parietal eminence* are the guiding landmarks by help of which the surgeon can often operate without marking down lines on the scalp. In children the fissure is sometimes higher and more oblique.

Reid's method of finding the fissure of Sylvius is to "draw a line from

a point 1¼ inches behind the external angular process to a point ¾ inch below the parietal eminence. The ascending branch starts from a point ¾ inch back from the anterior end of this line, and 2 inches (5 cm.) back of the external angular process."

IV. To outline the parieto-occipital fissure, find the lambda, mark a point 3 mm. anterior to it, draw a line through this at right angles to the longitudinal fissure, extending about 2.5 cm. (1 inch) on each side of the median line. This marks the position of the fissure. If the lambda cannot be felt, its position may be found by measuring the naso-occipital arc and taking 22.8 per cent. of it. This indicates the distance of the lambda from the inion or external occipital protuberance. The average

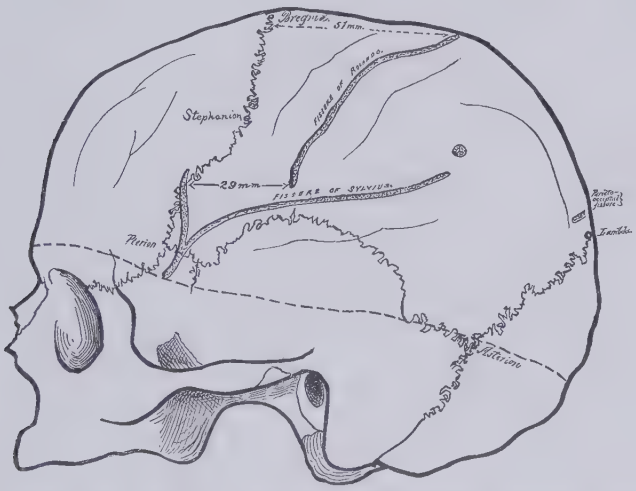


FIG. 257.—Showing the position of the bony points on the cranium, the sutures, and the principal underlying fissures, also the basal outline of the brain.

distance in male adults is 7.42 cm. (2⅞ inches). It is greater in women than in men by a little over a millimetre.

V. To outline the frontal lobes: The anterior end of the frontal lobes reaches to a point determined by the thickness of the frontal bone. This ranges from 2 to 8 or more mm. (1/12 to ⅓ inch). The floor of the anterior fossa reaches in front to a level a little above the supra-orbital margin (16 mm., ¾ inch—Heftler). It slopes down and backward, its posterior limit being indicated by the lower end of the coronal suture.

VI. To outline the temporal lobe and the lower border of the cerebrum: The temporal lobe is limited above by the fissure of Sylvius, below by the contour line of the lower border of the cerebrum. This latter corresponds to a line drawn from a point slightly (about 12 mm.) above the zygoma and the external auditory meatus to the asterion, and continued

on along the superior occipital curve to the inion. The anterior border of the lobe corresponds to the posterior border of the orbital process of the malar bone.

The temporal lobe is about 4 cm. (1⅝ inches) wide at the external auditory meatus. A trephine, as Bergmann states, placed half an inch above the meatus would enter the lower part of the lobe. The middle of the lobe is in a vertical line from the posterior border of the mastoid process. A line from the upper end of the fissure of Rolando to the point of the process would pass through this important sensory area (Fig. 259).

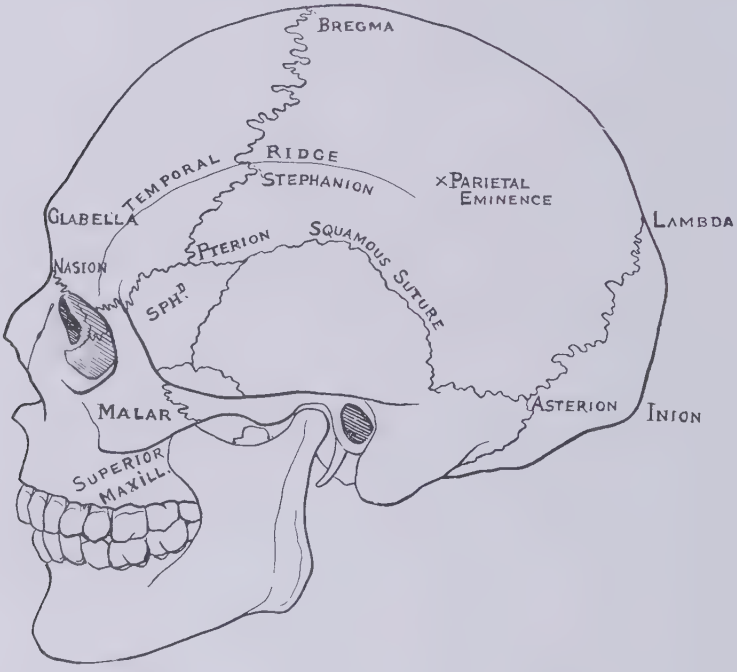


Fig. 258.—Showing anatomical landmarks on skull.

VII. To find the position of the central ganglia, viz., corpus striatum and optic thalamus, draw a line from the upper end of the fissure of Rolando to the asterion, practically a vertical line. This limits the optic thalamus posteriorly. A vertical line parallel to the first, a little in front of the beginning of the fissure of Sylvius, limits the corpus striatum anteriorly. A horizontal plane 45 mm. (1¾ inches) below the surface of the scalp at the bregma limits the ganglia superiorly. The ganglia lie about 35 mm. (1⅜ inches) below the superior convex surface of the brain (Féré).

VIII. To reach the lateral ventricles: A number of routes may be

COMPOSITE PHOTOGRAPH SHOWING RELATIONS OF CRANIAL SURFACE TO THE FISSURES AND CONVOLUTIONS. (*Alec Fraser.*)

taken. The lateral is recommended by Keen. Mark a point 1¼ inches behind the external auditory meatus and 1¼ inches above a base line made by drawing a line through the lower border of the orbit and the external auditory meatus. Trephine at this point and plunge the director into the brain in the direction of a point 2½ to 3 inches vertically above the opposite external meatus. The ventricle lies at a depth of 2 to 2¼ inches (5 to 5.7 cm.).

Mr. Alec Fraser has devised a way of mapping out the fissures by means of a series of composite photographs, so taken as to show the relation of the underlying parts to certain tapes tacked upon the skull. One of his figures is reproduced here (Plate IV).

In applying this method the surgeon tacks the tapes on the shaved scalp. Then looking at the diagram, he finds where the point in the brain is that he wishes to reach and notes its relation to the median lateral circumferential tapes. Then as the circumference of the illustrated head is to the circumference of the living one, so is the position of the area on the tapes in the illustrated head to the desired position of the same area in the living one. The illustration is a composite of several adult heads varying in circumference from 20¼ to 23½ inches.

The cranial measurements necessary to indicate the location of the important fissures and convolutions are given above. There are a number of other methods, but no very important improvements upon the original one of Reid. I have repeatedly verified the methods given and see no reason to change them. Some surgeons, however, prefer the Anderson-Makin method. According to this, the upper end of the fissure of Rolando corresponds to a point one-half to three-quarters of an inch behind the mid-point between the inion and glabella. The upper extremity of the parieto-occipital fissure corresponds to a point one-quarter of an inch behind the mid-point between the line of the Rolandic fissure and the inion.

As surgical approach to brain tumors is now done by large bone flaps, Dr. Charles K. Mills has outlined the various areas to be attacked, corresponding to the pre-frontal, post-frontal, parieto-temporal, mid-frontal, parietal and occipital areas. Having determined the location of the Rolandic, Sylvian and parieto-occipital fissures, the location of these flaps is easily determined.

APPENDIX

THE FUNCTION AND INNERVATION OF THE MUSCLES

Muscles of Tongue, Palate and Pharynx

Name of Muscle	Normal Function	Symptoms of Deficient Action	Innervated by	Represented in	Diseases in which Muscle is commonly Involved
Genio-glossus.	Pushes tongue to opposite side.	Tongue when protruded deviates to paralyzed side.	The twelfth nerve (hypoglossal).	Medulla.	
Stylo-glossus.	Raises tongue backward and upward.	Tongue cannot be moved backward or hollowed out (action deficient in many healthy subjects).	The twelfth nerve.	Medulla.	Bulbar palsies (acute and chronic); in specific and tuberculous diseases of base; dystrophies (rare).
Lingual muscle proper.	All movements of the tongue itself.	When lying in mouth deviation to healthy side; when protruded deviates to paralyzed side; if one or both halves are atrophied tongue looks shrivelled.	The twelfth nerve	Medulla.	
Azygos uvulæ.	Shortening of uvula.	Uvula deviates toward sound side; if both sides are paralyzed there are nasal tone and regurgitation through nose.	Ninth and tenth nerves.	Medulla.	As above.
Levator palati.	Raises the velum palati.	Arch cannot be raised in the intonation of "ah;" if paralysis is bilateral flapping of arch and regurgitation of food through nose.	Ninth and tenth nerves.	Medulla.	As above; see also seventh-nerve affections.
Palato-pharyngeal muscles.	Prevent food from passing toward upper part of pharynx and posterior nares.	Regurgitation of food; nasal speech.	Ninth and tenth nerves.	Pons.	Basilar affections.
Stylo-pharyngeus.	Helps to draw larynx upward so as to be closed by epiglottis and overtopped by tongue.	Imperfect deglutition; food gets into windpipe.	Glosso-pharyngeal.	Medulla.	Bulbar affections and diseases of the base.
Constrictors of pharynx.	Help to push food into gullet.	Food is swallowed very imperfectly; sticks in throat.	Pharyngeal plexus.	Medulla.	Diseases of the base (bulbar).
Laryngeal muscles.	Movements of vocal cords in respiration and in articulation.	Hoarseness and difficulty in breathing; laryngoscopic examination reveals false position of vocal cords (see special text-books).	Recurrent laryngeal nerve excepting the crico-thyroid muscle.	Medulla.	Bulbar troubles (similar symptoms may be caused by tumors and foreign bodies in larynx).

Muscles of Head and Neck

Sterno-cleido-mastoid.	Raises and turns face to opposite side, head inclines to same side; if both muscles act conjointly head is brought forward.	Inability to raise head from bed, or other horizontal position, if both muscles are affected; if one muscle is affected, no marked change of position, unless opposite muscle is contractured; spasm of muscle frequent; head inclined to one side.	Spinal accessory.	Medulla and second and third cervical segments.	In bulbar and cervical-cord affections; in later stages of progressive muscular atrophies; occasionally in neuritis.
Rectus capitis anticus major.	To flex head.	Cannot flex head so as to bring chin on chest.	Upper cervical.	Upper cervical segments.	Diseases of the cervical cord (myelitis, meningitis, tumor; progressive wasting of muscles).
Rectus capitis anticus minor.	To flex head.	Deficient rotation scarcely noticeable, unless sterno-cleido-mastoids are diseased.			

MUSCLES OF HEAD AND NECK.—*Continued*

Name of Muscle	Normal Function	Symptoms of Deficient Action	Innervated by	Represented in	Diseases in which Muscle is commonly Involved
Rectus capitis lateralis.	Slight rotation.	Deficient rotation scarcely noticeable, unless sterno-cleido-mastoids are diseased.	Upper cervical.	Upper cervical segments.	Diseases of the cervical region (myelitis, meningitis, tumor; progressive wasting of muscles).
Scaleni anterior medius, et posterior.	Elevate ribs when vertebral column is fixed; aid in inspiration; slight lateral flexion.	Deficient inspiratory movements.	Lower cervical nerves.	Upper cervical segments.	
Longus colli.	Flexion of vertebral column.	Imperfect flexion of upper spine.	Lower cervical nerves.		
MUSCLES OF SHOULDERS AND UPPER EXTREMITY					
Trapezius. 1. Clavicular portion (respiratory; outer third of clavicle to occipital bone).	Pulls head backward; rotates slightly toward side of muscle, so that chin is turned to opposite side; contraction of both clavicular portions bends head backward; slight elevation of shoulders; aids in deep inspiration.	Deficient backward movement of head; not marked as a rule because deep muscles perform this function; shoulder does not move during inspiration.	Spinal accessory.	Medulla and second and third cervical segments.	Progressive muscular wasting; diseases of medulla and upper cervical cord; clavicular portion least frequently involved.
2. Middle portion (from acromion and outer spine of scapula to ligamentum, nuchæ and upper dorsal spines).	Raises shoulder-blade; elevation of acromion (clavicle goes along).	Acromion depressed by weight of upper extremity; inner upper angle may be pulled upward by levator anguli scapulæ; internal lower angle is nearer to median line.	Spinal accessory nerve.	As above.	As above.
3. Lower portion and adductor.	Adduction of scapula toward median line.	Margin of scapula is about 10 cm. distant, instead of being 5 or 6 cm. distant from median line; loss of adductor may be covered up by action of rhomboids; rounding of back.	Spinal accessory nerve.	Medulla and second and third cervical segments.	
Rhomboids.	Oblique movement of scapula from below, upward and inward, so that inferior angle is brought nearer the median line; hold spinal margin of scapula down to thorax.	Deep groove between inner margin of scapula and thorax; if serratus is normal, this groove disappears if arm is extended forward; shoulder-blade cannot be approximated to median line. (According to Duchenne this can be effected by upper portion of latissimus dorsi.)	Fifth cervical.	Fourth and fifth cervical segments.	As above.
Levator anguli scapulæ.	Draws superior inner angle of scapula upward; aids in shrugging of shoulders.	Isolated paralysis rare.	Third and fifth cervical nerves.	Second and fourth (?) cervical segments.	Dystrophies and cervical diseases.

MUSCLES OF SHOULDERS AND UPPER EXTREMITY.—*Continued*

Name of Muscle	Normal Function	Symptoms of Deficient Action	Innervated by	Represented in	Diseases in which Muscle is commonly Involved
Serratus magnus.	Rotation of shoulder-blade outward, and slight elevation of acromion; holds inner margin of scapula to thorax; brings arm from horizontal to vertical position.	Scapula pulled upward; lower inner angle nearer the median line; arm cannot be raised above horizontal position; if arm is stretched forward scapula is removed from thorax ("winged scapula"); during abduction of arm, scapula is moved nearer to median line, and crowds trapezius and rhomboids forward.	Posterior thoracic nerve.	Fifth and sixth cervical segments.	Progressive muscular atrophies (dystrophies); neuritis of part of the brachial plexus; after traumatic injuries to shoulder; in cervical-cord affections.
Deltoid (three divisions).	To raise arm to horizontal position and forward, outward or backward; movements possible only if scapula is fixed by action of serratus and trapezius.	Can raise shoulder but not arm; shoulder flattened (atrophy); groove between acromion and head of humerus; each division of deltoid may be paralyzed singly.	Circumflex.	Fourth, fifth and sixth cervical segments.	As above; also in Erb's form of obstetrical paralysis.
Infra-spinatus. Teres minor.	Rotator humeri posticus (Duchenne); rotate arm outward.	Arm cannot be moved outward. Difficulty in writing (Duchenne).	Supra-scapular. Circumflex.	Fourth, fifth and sixth cervical segments.	As in case of deltoid.
Subscapularis.	Rotator humeri anticus (Duchenne); rotates arm inward.	Arm cannot be moved inward; scapula is rubbed against ribs.	Subscapular nerve.		
Supra-spinatus.	Helps to steady shoulder-joint and to elevate arm forward and outward; outer angle of scapula is depressed.	According to Duchenne, humerus is separated still farther from acromion, if supra-spinatus is affected in addition to deltoid.	Supra-scapular.	Fourth cervical.	As above.
Latissimus dorsi.	Pulls the arm when raised downward and backward; if arm is at rest upper portion brings scapula nearer the median line; united action of upper third of both muscles causes extension of dorsal trunk; single action causes lateral movement of trunk.	Arm cannot be moved backward; insufficient extension of dorsal spine; trunk cannot be moved laterally.	Subscapular, also branches of dorsal and lumbar nerves passing through muscle.	Sixth and seventh cervical.	As in progressive atrophies and dystrophies in cervico-dorsal lesions; in neuritis.
Teres major.	Rotates raised humerus inward; adduction of arm to thorax; slight elevation of shoulder.	Very few symptoms; action supplied by other muscles.	Subscapular.	Seventh cervical.	As above.
Pectoralis major.	Clavicular portion depresses humerus from raised position to horizontal; adduction of arm, as in giving a blessing; sternal portion depresses arm completely, and if arm is at rest draws acromion forward and backward.	Imperfect abduction of arm; paralysis can be discovered best by extending arms and trying to press volar surfaces against each other.	Anterior thoracic.	Fifth, sixth and seventh cervical.	Amyotrophies and dystrophies, chiefly; also in lesions of brachial plexus.

Muscles of Arm, Forearm and Hand

Name of Muscle	Normal Function	Symptoms of Deficient Action	Innervated by	Represented in	Diseases in which Muscle is commonly Involved
Triceps.	Extends forearm; long head of triceps, and coraco-brachialis help to keep head of humerus in position.	Arm cannot be extended except by its own weight; if long head of triceps is affected subluxation of head of humerus occurs easily.	Musculo-spiral.	Sixth, seventh, eighth cervical segments.	Poliomyelitis and other affections of cervical cord; traumatic injuries; amyotrophies and dystrophies (triceps escapes in many peripheral palsies).
Biceps.	Flexion and supination of forearm.	Flexion deficient, but can be carried out in part by other muscles.	Musculo-cutaneous.	Fourth, fifth, sixth cervical.	
Supinator longus.	Flexes forearm and aids in pronation.	Flexion and pronation deficient; muscle does not stand out prominently if arm is flexed and attempt is made by another to extend if forcibly; if muscle is atrophied arm is spindle-shaped.	Musculo-spiral.	Fourth, fifth cervical.	As above; involved in peripheral neuritis (traumatic), not in lead palsy.
Supinator brevis.	Supinates hand when forearm is extended.	Deficient supination of hand.	Musculo-spiral.	Fifth cervical.	Diseases as above; also in peripheral palsies.
Extensor carpi radialis longus et brevis.	Extension and abduction of wrist; the shorter muscle has pure extension action only.	Wrist cannot be flexed dorsally (extended) or abducted; flattening of forearm.	Musculo-spiral.	Seventh cervical.	As before; especially in neuritis.
Extensor carpi ulnaris.	Extension and abduction of wrist.	Wrist cannot be flexed dorsally or adducted; "drop-wrist" is characteristic of paralysis of extensors.	As above.	Seventh cervical.	As above.
Extensor digitorum communis.	Extension of first phalanges of all fingers and abduction.	First phalanges cannot be extended nor fingers abducted; grasp is weak because flexor muscles are shortened and cannot contract forcibly.	Musculo-spiral.	Seventh cervical.	As above.
Extensor indicis.					
Extensor minimidigiti					
Flexor carpi radialis.	Flexion of wrist and pronation.	Deficient flexion.	Median.	Eighth cervical.	As above.
Flexor carpi ulnaris.	Flexion of wrist and supination.	Flexion and supination impaired.	Ulnar.	Eighth cervical.	As above.
Palmaris longus.	Flexion of wrist only.	Flexion impaired; no anomalous position of hand from paralysis of wrist as hand falls by its own weight; the flexors of fingers may act as substitutes.	Median.	Eighth cervical.	As above.
Flexor digitorum sublimis.	Flexes second phalanx toward first.	Second phalanx cannot be flexed.	Median.	Eighth cervical.	As above.
Flexor digitorum profundus.	Flexes last two phalanges toward first.	Last two phalanges cannot be flexed.	Ulnar and median.	Eighth cervical.	As above; muscle should be tested with special care in cases of traumatic injuries.
Interossei and lumbricales.	Abduction and adduction of fingers if first phalanges are extended; flexion of first phalanges and simultaneous extension of second and third phalanges.	Fingers cannot be abducted or adducted; interosseous spaces are very marked; "Main en griffe" due to extension of first phalanges and flexion of second and third phalanges.	Ulnar, which also supplies third and fourth lumbricales; median supplies first two and sometimes third lumbricales.	Eighth cervical, first dorsal.	As above; often the first muscles to be affected in progressive spinal atrophies.

MUSCLES OF ARM, FOREARM AND HAND.—*Continued*

Name of Muscle	Normal Function	Symptoms of Deficient Action	Innervated by	Represented in	Diseases in which Muscle is Commonly Involved
Thenar muscles.	Extends first phalanx and abducts metacarpal bone; acts with adductor pollicis longus.	Impairment of extension and abduction; flattening of ball of thumb.	Musculo-spiral.	First dorsal.	As before; more especially in amyotrophies and neuritis.
Extensor pollicis brevis.	Extends both phalanges of thumb; also adduction of metacarpal bone and backward movement of thumb.	Deficient extension and abduction; second phalanx is flexed toward first.	Musculo- spiral.	First dorsal.	As above.
Abductor pollicis longus.	Abduction of metacarpal bone; aids in flexion of hand.	Deficient abduction of metacarpal bone; if this muscle and extensor pollicis brevis are paralyzed adduction results.	Musculo-spiral.	First dorsal.	As above.
Abductor pollicis brevis.	Opposition of thumb.	No opposition movement.	Musculo-spiral. Median.	First dorsal.	As above.
Opponens pollicis and outer portion of the flexor brevis.					
Abductor pollicis brevis flexor brevis and adductor.	Flex first phalanx and extend second phalanx (like interossei), also have an abduction and adduction action.	No flexion; if muscles are paralyzed and atrophied, ape hand is formed.	Median and ulnar.		As above.
Flexor pollicis longus.	Flexes end phalanx.	No flexion of end phalanx.	Median.		As above.

Muscles of Back and Lower Extremities

Name of Muscle	Innervated by	Symptoms of Deficient Action
Erector spinæ; sacro-lumbalis longissimus dorsi.	Dorsal nerves. Second to twelfth dorsal segments.	Lordosis of lower spine; perpendicular line from shoulder falls behind os sacrum; unilateral palsy causes deflection of spine toward sound side.
Abdominal muscles.	Dorsal nerves. Second to twelfth dorsal.	Lordosis with protrusion of nates and abdomen; other actions deficient; cannot straighten up from recumbent position without assistance of hands.
Quadratus lumborum.	Lumbar nerves.	Lateral movements of lower vertebræ imperfect.
Adductor muscles.	Obturator nerve, great sciatic and crural.	No adduction; thigh rolls outward.
Sartorius.	Crural. Third lumbar segment.	Flexion impaired; acts imperfectly.
Quadriceps femoris.	Crural. Third lumbar.	Leg cannot be extended; to test it ask patient, who is lying down with hip bent, to stretch out the leg; when patient is sitting down to extend leg.
Ilio-psoas.	Crural (lumbar plexus). Fourth lumbar.	Flexion difficult; in bed thigh cannot be flexed; difficulty rising from the horizontal position.
Tensor fasciæ latæ.	Superior gluteal. Fourth lumbar.	
External rotators: Pyriformis, Gemelli, Quadratus femoris.	Sacral plexus (muscular branches). Fifth lumbar.	Deficient outward rotation; leg turned inward.
Internal obturator. External obturator.	Obturator nerve (lumbar plexus).	
Gluteal muscles.	Inferior gluteal (sacral plexus). First and second sacral.	No extension of thigh; great difficulty in climbing; no abduction of thigh; waddling gait, exaggerated movement of plevis.
	Gluteal superior. First and second sacral.	
Biceps: semitendinosus and semimembransous.	Sciatic. Fifth lumbar segment.	Deficient flexion; action of quadriceps may cause excessive extension; in standing thigh is flexed to excess; trunk moved backward.
Gastrocnemius (also plantarius and soleus).	Internal popliteal. Fifth lumbar.	Deficient flexion of foot; heel cannot be raised; cannot stand on tiptoes.
Anterior tibial muscles (tibialis anticus, extensor digitorum, and extensor pollicis longus).	Anterior tibial. Fifth lumbar and first sacral.	Deficient extension; "drop-foot," toes scrape floor; to clear this, excessive flexion at knee and hip; contracture of flexors and pes equinus or equinovarus.
Peroneus longus.	Peroneal. First and second sacral segments.	Deficient abduction; plantar arch lessened; increased by contracture.
Posterior tibial muscle	Posterior tibial nerve. First and second segments.	Flatfoot; walking tiresome. Deficient abduction or adduction; deformities result from deficiencies.
Peroneus brevis.	Peroneal. First and second segments.	
Interossei pedis et lumbricales.	Posterior tibial. First and second segments.	Abduction and adduction of toes deficient; paralysis of interossei; hyperextension of first phalanges; second and third flexed (clawed foot).
Adductor; flexor brevis and abductor hallucis.	Posterior tibial. First and second segments.	Deficient flexion of toes; foot cannot be pushed off ground easily.

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